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PROBLEMS OF MULTIPLE TRANSFUSIONS, Frank H. Gardner, M.D., Boston . . . 93

PAROTID SIALOGRAPHY—Roentgenologico-Surgical Correlation in a Series of 70 Cases, Robert A. J. Einstein, M.D., and Samuel L. Perzik, M.D., Beverly Hills . 98

ORAL ADMINISTRATION OF ORINASE® (TOLBUTAMIDE)—Clinical Observations of Effect on Nondiabetic and Diabetic Humans, Thomas H. Lambert, M.D., William F. Bethard, M.D., Solon Palmer, Jr., M.D., and Lee S. Monroe, M.D., La Jolla . 103

PROSTHETIC ARTERIES—Resection of the Abdominal Aorta and Replacement with Plastic Materials, Arthur C. Miller, M.D., Arnold A. Michals, M.D., and Raymond G. Auvil, M.D., Loma Linda 109

MALIGNANT CARCINOID TUMOR—Altered Reactivity of the Skin to Serotonin in Two Patients, Maurice Rotbart, M.D., Long Beach 112

STAPES MOBILIZATION FOR OTOSCLEROTIC DEAFNESS—The Monitored Peribasal Technique, Victor Goodhill, M.D., Los Angeles 114

THE TREATMENT OF DIABETES MELLITUS, William W. H. Pote, Jr., M.D., Los Angeles 123

RECENT DEVELOPMENTS IN SURGICAL SKIN PLANING, Samuel Ayres, III, M.D., and J. Walter Wilson, M.D., Los Angeles, and Ralph Luikart, II, M.D., Santa Barbara 127

ALCOHOLISM—The Chronological Background Leading to California's Present Program, Wendell R. Lipscomb, M.D., Berkeley 133

GALLSTONE INTESTINAL OBSTRUCTION, Frank A. Rogers, M.D., Whittier, and Richard Carter, M.D., Los Angeles 140

ABSENCE FROM SCHOOL—Its Relationship to Illness in the Community, Arthur H. Parmelee, Jr., M.D., and Bella F. Schimmel, M.D., Los Angeles 144

PREPARTUM NAUSEA AND VOMITING—A New Approach to Treatment, William G. Caldwell, M.D., and Kenneth Hobbs, M.D., Los Angeles 149

BURSITIS—OFTEN AN INEXACT DIAGNOSIS, Donald P. Carpenter, M.D., Riverside 151

MARGINAL PEPTIC ULCER IN ELDERLY PATIENTS, Clarence E. Stafford, M.D., and David B. Hinshaw, M.D., Los Angeles 155

EMPLOYMENT OF PERSONS WITH EPILEPSY AND HEART DISEASE, Lloyd T. Lorbeer, M.D., and Charles I. Barron, M.D., Burbank 160

CASE REPORT:

Acute Barbiturate Intoxication—A Case with Extremely High Content in the Blood and Protracted Coma with Recovery, Richard I. Crone, Colonel (MC), David E. Johnson, Captain (MC), and Eric I. Anderson, Captain (MC), U. S. Army 166

EDITORIAL, 170 • CALIFORNIA MEDICAL ASSOCIATION, 173 • WOMAN'S AUXILIARY, 182
NEWS AND NOTES, 183 • BOOK REVIEWS, 191

C.M.A. Annual Meeting, Los Angeles, April 27 to April 30, 1958
(For Announcement and Hotel Reservations, see pages 178-179)

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Problems of Multiple Transfusions

FRANK H. GARDNER, M.D., Boston

• **The use of blood infusion in large amounts is increasing sharply. Increased knowledge of blood group antigens has alerted physicians to the possible hazards of hemolytic reactions to subgroups that must be eliminated by proper cross-matching techniques. Multiple transfusions of preserved blood often defeat their purpose in control of bleeding, for thrombocytopenia is enhanced. Careful selection of blood or preparations of plasma concentrates offer increased protection to the recipient.**

Plastic bag equipment increases the yield of viable platelets and keeps blood in usable condition for longer periods of storage. The use of multiple transfusions has complicated the selection of preserved blood to control pigment metabolism.

DURING THE PAST DECADE there has been a sharp increase in the use of whole blood and plasma components. The vigorous orientation toward rehabilitation of patients with tumors, the greater use of corrective vascular operations and maintenance of comfort in anemic patients have required more transfusions. The expansion of blood requirements in a small teaching hospital are plotted in Chart 1. In 1956, some 4,585,000 units of blood were used in the United States, and even greater needs may be anticipated in the next decade.

The need for multiple transfusions has initiated a remarkable experiment in immunization. The blood group antigens may immunize the recipient as a result of a single transfusion, and future transfusions may become dangerous. Almost 50 blood group factors are known and new factors certainly will be defined. Many of these antigens cause only mild reactions. However, despite the routine accepted laboratory cross-matching procedures for ABO group compatability, over 90 per cent of the transfusions are potentially sensitizing to the recipient.[5] For the most part physicians can protect patients during multiple transfusion therapy if the blood is adequately screened in the laboratory with the techniques available.

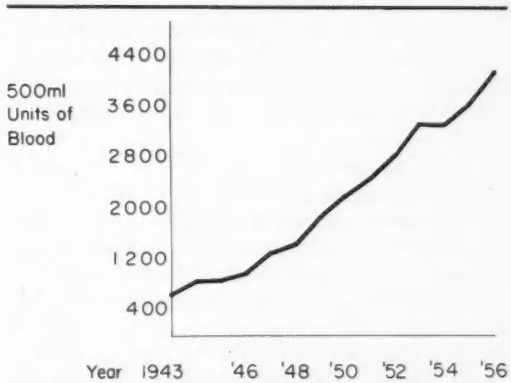

Chart 1.—Use of whole blood in a 260-bed teaching hospital in a period of 13 years (Peter Bent Brigham, 1943-1956). The massive increase in transfusions probably is best related to requirements for vascular operations.

Guest Speaker's Address: Presented before a Joint Meeting of the Sections on General Surgery and Internal Medicine at the 86th Annual Session of the California Medical Association, Los Angeles, April 28 to May 1, 1957.

Assistant Professor of Medicine, Harvard Medical School, and Senior Associate in Medicine, Peter Bent Brigham Hospital, Boston.

From the Richard C. Curtis Hematology Laboratory, Peter Bent Brigham Hospital and Department of Medicine, Harvard Medical School, Boston.

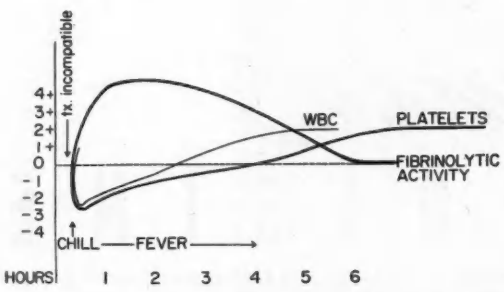

Chart 2.—Reactions in peripheral blood following mismatched transfusion reaction (tx incompatible). The increased plasma fibrinolytic activity is associated with decreased circulating fibrinogen levels. The transient placelet depression will increase bleeding tendency. Modified from Stefanini and Dameshek.[10]

When a patient has massive bleeding, adequate selection and cross-matching procedures may not be feasible. The physiologic response to incompatible blood may enhance the bleeding state. Following the infusion of mismatched blood, the platelet count declines and transient leukopenia occurs (Chart 2). Fibrinolytic activity of the plasma is activated to decrease the circulating fibrinogen level and augment dissolution of recent thrombi necessary for hemostasis.[10] Excessive oozing from tissues may alert the surgeon that a transfusion reaction exists, for anesthesia may mask the chill and fever. The bleeding tendency is increased further by the febrile response and vasodilatation. This reaction persists for several hours and may increase the blood replacement requirements. When possible, an excess of blood should be cross-matched as a safeguard against the hazard of infusing blood without proper safeguards. Patients who have had previous transfusions should be cross-matched with macromolecular substances or evaluated with antihuman globulin rabbit (Coombs) serum to exclude "incomplete" circulating red cell antibodies in the recipient. As a routine policy, in the absence of Rh-negative typing, all recipients who have received five transfusions are cross-matched with Coombs serum for antibodies.

Multiple infusions of stored bank blood collected in National Institute of Health formula A or B, acid citrate dextrose anticoagulant solution will not contain viable platelets. Excessive loss of blood and replacement with bank blood allows the platelets to be "washed out" of the peripheral circulation. Krevans and Jackson[8] correlated platelet counts of the peripheral blood with the number of units of blood infused. As blood replacement exceeds 10 units, thrombocytopenia may be a contributing factor to further bleeding. The circulating platelet levels are not always related to the amount of blood infused, but rather to the time interval in which the blood is administered. Hence, replacement of 15 units in an hour will cause thrombocytopenia, while the same amount of blood given over a period of several days may not affect the platelet count appreciably. As a precautionary policy, the blood bank personnel should alert the physician when 10 units of blood have been administered over a short interval, in order that fresh blood drawn within the hour containing viable platelets may be used. Thrombocytopenia has occurred most frequently, in my experience, during operations on the great vessels and blood replacement in patients with esophageal varices. The bone marrow does not have the capacity to replace or maintain the circulating level of blood platelets rapidly. Craddock and associates[3] demonstrated in dogs the slow recovery of the blood platelet levels after plasmapheresis brought about severe thrombocytopenia. A recovery period of three to five days was necessary for the bone marrow to achieve the pretreatment platelet level. Similar recovery periods have been observed clinically after infusion of 15 to 25 units of banked blood. In essence, a duplication of Craddock's studies may be observed by the infusion of banked blood without viable platelets. The bone marrow does not have a large reservoir of platelets available, as is the case with leukocytes, to restore the circulating platelet levels after depletion by massive bleeding.

Patients with bleeding esophageal varices and portal hypertension need special attention for blood replacement. It many instances moderate thrombocytopenia associated with splenomegaly may have existed before the bleeding episode. These patients appear to have a delayed activation of platelet response to bleeding, a phenomenon that has suggested bone marrow inhibition. Hence, one may not anticipate a rapid recovery of the peripheral platelet values after severe bleeding.[4] Patients with thrombocytopenia undergoing portacaval shunts for portal hypertension should receive platelet concentrates before operation to lessen transfusion requirements and to correct thrombocytopenia. If active bleeding prevents preparation of the patient in this way, the surgeon must recognize that thrombocytopenia may develop more rapidly after the rapid infusion of five to ten units of bank blood. Although such surgical procedures may be begun with preserved blood used at first, fresh whole blood collected in plastic bags should be used after the first few transfusions to assure adequate viable platelets.[6]

The use of bank blood for massive transfusions also carries the risk of thrombocytopenia due to plasma factors. Stefanini[11] demonstrated that stored plasma will depress circulating platelet levels by from 20 to 40 per cent.[11] The platelet depression is

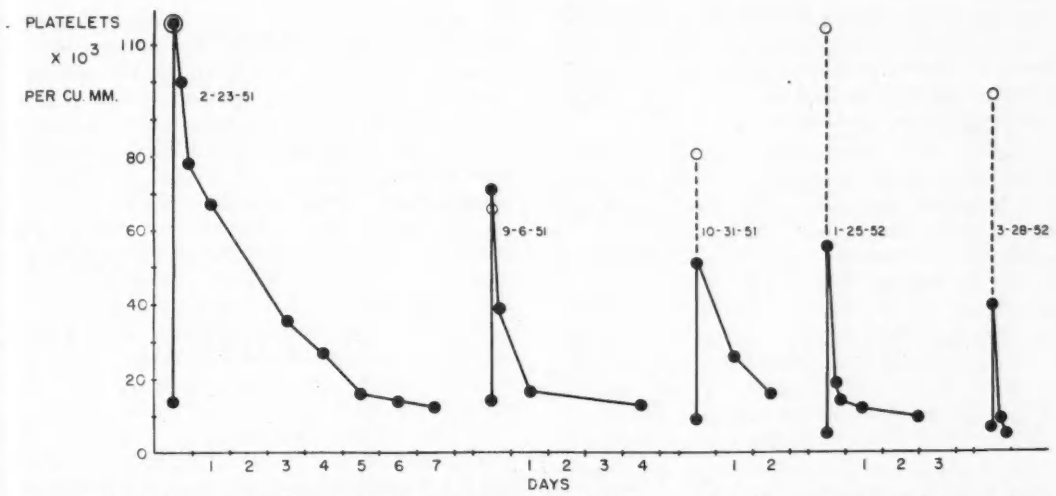

Chart 3.—Evidence of platelet sensitization in patient with aplastic anemia receiving many transfusions. Administration of fresh blood showed initially normal platelet survival of five to seven days. Gradually the survival period decreased and only a temporary appearance of platelets was measured in the patient after transfusion. The dotted lines indicate the theoretical platelet count that was anticipated. The progressive decrease in yield after transfusion suggests immediate platelet destruction as immunization developed.[6]

transient; it exists for a few hours. However, human stored or reconstituted plasma, infused as an adjunct to control hypotension, may well enhance bleeding. This complication may be of clinical importance in a severely burned patient who already has thrombocytopenia related to the injury and needs plasma protein replacement.[9] This transient platelet depression has not been observed when human serum albumin is used, which emphasizes the value of this purified material as a plasma expander.

In Table 1 the viable platelet yield of blood transfused by various procedures is shown. The numerical values for platelets in the in vitro preparation are not important, *per se*, but rather the recovery or yield of circulating (clot promoting) platelets obtained in the recipient should be considered the end point of effectiveness. A sensible program of advanced planning will prevent thrombocytopenia. A patient who has received numerous units of bank blood should have frequent determination of bleeding time. This simple test appears to be the best guide for the sum-total of vascular and plasma factors that influence clotting during multiple transfusions. A nonanemic patient with nonimmune thrombocytopenia should be protected with platelet concentrates. The hemorepellent surfaces offered by plastic bag equipment and tubing has improved the efficiency of preparing platelet suspensions, and good design has helped to insure sterility during the manipulations of centrifugation and concentration. It is important that all recipient infusion sets have protective screen filters removed for the infusion of fresh blood or platelet preparations.

TABLE 1.—*Circulating Yield (Per Cent) of Viable Platelets in the Recipient After Various Transfusion Techniques. (Platelets Are Destroyed by Centrifugation and by Contact with Glass Surface.*[6])

Transfusion Procedure and Equipment	Per Cent of Platelets Viable
Direct, silicone multiple syringe	80 to 90
Fresh (1 hour), plastic bag (ACD/EDTANa_2)	75 to 80
Platelet-rich plasma, plastic bag	60 to 65
Platelet concentrates, plastic bag	50 to 55
Fresh gravity collection, glass vessel	15 to 20
Stored blood, glass vessel (24 hours)	0

Many patients with aplastic anemia, leukemia, nephritis and carcinomatosis have recurring problems of multiple transfusions. Since the procedures in such circumstances are elective, the physician may be assured of compatibility of red cell antigens. However, no satisfactory techniques are available for typing of platelets and leukocytes. The value of blood administered for platelet replacement diminishes and eventually the platelet yield in the recipient is insufficient to protect the patient.[7,12] Transfused platelets may be followed in the recipient for five to seven days. However, if previous transfusions have immunized the patient, the infused platelets may disappear within minutes (Chart 3). The rapid destruction of platelets may be associated with flushing, tachycardia and fever. A similar immune response may be observed following leukocyte sensitization from multiple transfusions. In addition to high fever, severe hypotension and cyanosis may occur, and transient pulmonary infiltrates may be

observed in x-ray films.[1] These findings suggest that the leukocytes are agglutinated by white cell antibodies in the recipient from previous transfusions, and that the agglutinated leukocytes are trapped in the pulmonary capillaries.

The severity of these reactions may be altered by centrifuging whole blood and removing the buffy coat before the transfusion. Washing the red cell mass with saline solution will adequately remove leukocytes and platelets from blood to be given to a highly sensitized recipient.[2] In retrospect, probably many febrile reactions classified previously as "plasma protein reaction" in reality were immune responses to leukoagglutinins. If a physician elects to give multiple transfusions, only the red cells should be infused. Removal of the plasma and buffy coat will lessen sensitization of the recipient.

Little interest has been directed toward problems of clinical citric acid intoxication during the transfusion of citrated blood. However, elevated blood citrate levels with intoxication may be seen in patients with liver disease, or during rapid or prolonged transfusions of bank blood. In most normal adults citrate toxicity will not appear until the infusion rate exceeds 5,000 ml. of citrated blood per hour. This rate is now seen frequently with severe bleeding problems (esophageal varices and vascular operations). A patient with impaired liver function or obstruction to the great vessels of the abdomen during vascular operations is liable to have citrate intoxication. Although depression of serum ionized calcium levels and elevated serum citrate levels may be observed, the mechanism for hypotension and altered electrocardiographic patterns may be associated with altered relationships between calcium and potassium ions in the extracellular fluid of the myocardium.[12] Decalcified blood collected from donors by passing across a cation exchange resin should be considered when multiple transfusions are given rapidly.[14] Accessory clotting factors are not altered in decalcified blood, and such transfusions are of real value for patients with severe liver disease. The rapid infusion of calcium-depleted blood will not depress circulating serum calcium levels and interfere with the blood coagulation system. Use of blood with calcium removed for replacement is more ideal than efforts to replace calcium salts parenterally when using citrated blood. If blood decalcified by cation exchange is not available, the hazards of citrate infusion may be controlled in part by more vigorous use of sedimented red cells with plasma expanders to limit the volume of citrated plasma.

Under present regulations most blood banks will retain blood collected in glass bottles with acid citrate dextrose (ACD) anticoagulant solution for 21 days. At the end of three weeks 70 per cent of the preserved red cells are viable after infusion into a recipient. The 30 per cent nonviable red cells are destroyed rapidly during the first 24 hours. If a patient receives only a few transfusions, this does not raise circulating pigment to hazardous levels—namely, about 60 ml. of red cells per unit of blood. If the bank blood has been stored for less than 21 days, the proportion of nonviable red cells will be smaller. If large quantities of blood are required to control bleeding, the physician should recognize that the recipient may have some embarrassment in handling blood pigments derived from the nonviable red cells in preserved blood. Improved viability of red cells may be obtained by collecting blood in plastic bags rather than glass bottles. Utilizing radioactive sodium chromate to evaluate the life span of red cells in recipients, Strumia[13] concluded that blood preserved in plastic bags at 5°C. for 21 days had 87 per cent viability in contrast to 70 per cent in standard glass bottles. Similarly, Walter and co-workers[15] noted 84 per cent survival of blood collected in plastic bags. This method of preservation offers additional protection to decrease the blood pigments that the recipient's reticuloendothelial tissues must handle when a large amount of blood (perhaps the entire blood volume) is replaced with massive transfusions of preserved blood.

The demand on the recipient to clear the plasma of red cell pigments is met in most instances. But the presence of liver disease prevents rapid reduction of plasma bilirubin; and hypotension, along with the associated renal arteriolar vasoconstriction, sensitizes the kidney's reaction to circulating oxyhemoglobin.[17] As an illustrative problem, a 52-year-old man recently underwent operation to correct aortic stenosis. The patient had congestive heart failure before operation but tolerated the procedure well, and the technical results were excellent. His blood was of Group A, Rh negative. Ten units of blood were infused and many of the bottles were close to the accepted expiration date—namely, 18 to 20 days. Because of the storage period, the patient received hemoglobin derived from 275 ml. of nonviable red cells. The stress of the procedure as well as embarrassed liver function resulted in ischemic nephrosis, which undoubtedly was more severe because of the circulating pigment (Chart 4). The patient could not accept or metabolize the destruction of the nonviable red cells and died; the result of a chemical death rather than a complication of operation. Here then is a problem that will need more careful attention in the future. Multiple transfusions should be planned, especially with elective operations, to use relatively fresh bank blood (less than five days old) to protect the patient from a

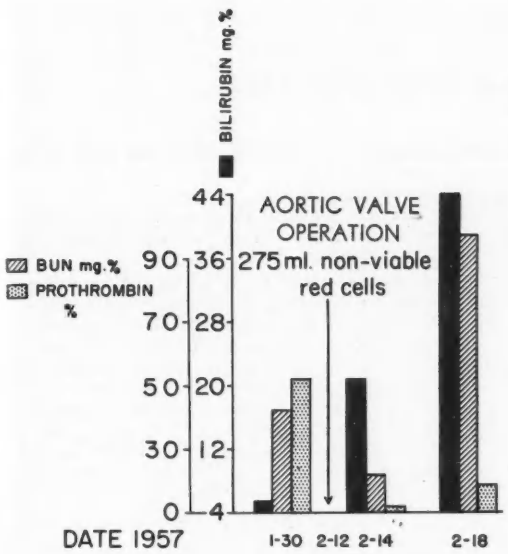

Chart 4.—Development of ischemic nephrosis following infusion of nonviable red cells from old bank blood (18 to 20 days). Progressive pigment retention was indicated by impaired renal excretion (elevated blood urea nitrogen: BUN) and inadequate liver function (depressed prothrombin per cent).

hazardous circulating blood pigment level. The presence of hypotension and poor liver function enhances the complication of acute tubular necrosis by the hemolyzed red cells. This is not a laboratory problem of cross-matching, but rather a "storage lesion" of preserved red cells.

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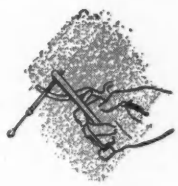


Parotid Sialography

Roentgenologico-Surgical Correlation in a Series of 70 Cases

ROBERT A. J. EINSTEIN, *M.D.*, and SAMUEL L. PERZIK, *M.D.*, Beverly Hills

• **A study was made of the correlation of sialographic, clinical and surgical observations in a series of 70 cases.**

Sialography is a useful tool in the differentiation between extrinsic and intrinsic space-occupying masses in the parotid region. It permits differentiation between inflammatory lesions and neoplasms, thus eliminating needless surgical explorations. It alerts the surgeon to masses involving the deep lobe. It can be easily performed and causes little discomfort.

Bearing in mind its limitations, sialography is an important diagnostic and preoperative aid in the management of parotid lesions.

THE MANAGEMENT of parotid tumors is hazardous and any diagnostic and therapeutic aid is desirable. Sialography—the roentgenographic visualization of the ductal system of the parotid gland—has been used since 1904, with techniques that have not changed much since that time. Although many papers have been written on the subject, no attempt has yet been made to evaluate the procedure by correlating clinical, roentgenological and surgical observations.

Material

Table 1 shows the final diagnosis in 70 consecutive sialographic studies. The age of the patients ranged from 14 to 79 years. All the roentgenographic studies were done by one radiologist (R. A. J. E.) and all operations by one surgeon (S. L. P.), thus eliminating many variables and errors of comparison.

Anatomy

The parotid gland is located below and in front of the ear and in the retromandibular fossa. It extends from the zygomatic arch down to a point a centimeter or two below the angle of the mandible. It has a deep and a superficial lobe. Accessory lobes, usually located anterior to the main portion of the parotid gland and along Stensen's duct, are not unusual.

The facial nerve, which is of great surgical importance, straddles the isthmus between the superficial and the deep lobe. Stensen's duct and its major radicles are situated deep to the branches of the facial nerve. Thus, visualization of Stensen's duct will give an indication of the approximate location of the facial nerve. Masses in the deep lobe cause lateral displacement of the major ducts and thereby of the facial nerve; depending on their location, masses in the superficial lobe occasionally produce anterior or posterior displacement of the duct system.

Technique

The injection of contrast medium into Stensen's duct is done on the x-ray table, without preparation. After a preliminary flat film is taken to rule out calculi or other abnormal soft tissue or osseous shadows, the opening of the duct is probed and gently dilated with a cannula of the kind used for the lacrimal duct. Between 0.5 and 1.5 cc. of Iodochlorol,® warmed to body temperature to lessen its viscosity, is then injected through a blunt needle (20 to 23 French) with a Luerlock syringe. The injection can be facilitated by pulling the cheek forward, thus straightening the duct. The injection is made with moderate pressure and the patient warned beforehand to expect some pain while the pressure is maintained. Local anesthesia to the duct opening or instillation of a small amount of an anesthetic agent into the duct does not lessen the slight and only temporary discomfort. The optimal amount of contrast medium to be injected can be gauged by the backflow welling from Stensen's opening during

TABLE 1.—*Final Pathological Diagnosis in a Series of Cases in Which Sialographic Studies Were Done*

Diagnosis	No. Cases
A. Neoplastic lesions:	
1. Mixed tumors	28
2. Muco-epidermoid tumors	8
3. Warthin's tumors	9
4. Angioma	1
5. Giant follicular lymphoma	1
6. Lymphoepithelioma of Godwin	1
7. Boeck's sarcoid	1
8. Metastatic melanoma	1
B. Nonspecific lymphadenitis	2
C. Cysts:	
1. Ductal	3
2. Sebaceous	1
D. Sialadenitis and sialangiectasis	2
Total	58

Presented before the Section on Radiology at the 86th Annual Session of the California Medical Association, Los Angeles, April 28 to May 1, 1957

*From the Departments of Therapeutic Radiology and Surgery (Head & Neck Clinic), Cedars of Lebanon Hospital, Los Angeles.

the injection. The needle is then withdrawn and pressure is applied by means of a piece of gauze, held in position by the tightly closed jaws. X-ray films are taken immediately in the postero-anterior and lateral projections. Stereoscopic views, mainly in the postero-anterior projection, have been found most helpful. Additional contrast medium can be injected if the duct is not inadequately filled. (This was not necessary in more than a few cases in the series.)

The entire procedure takes only about 20 minutes. Swelling of the gland usually persists for a few

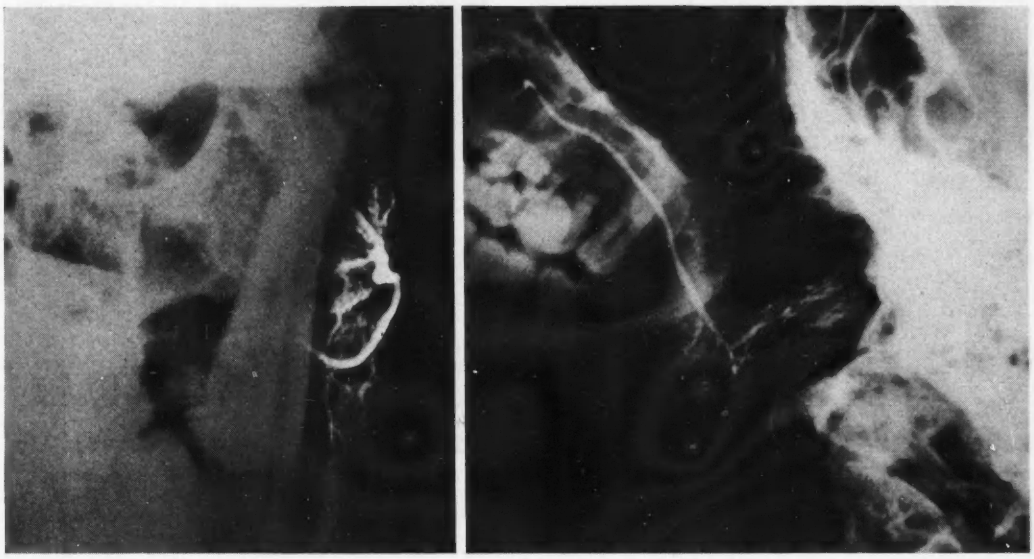


Figure 1.—Normal parotid gland. Evenly distributed "arborization" in all lobes. *Left:* Postero-anterior projection. *Right:* Lateral projection.

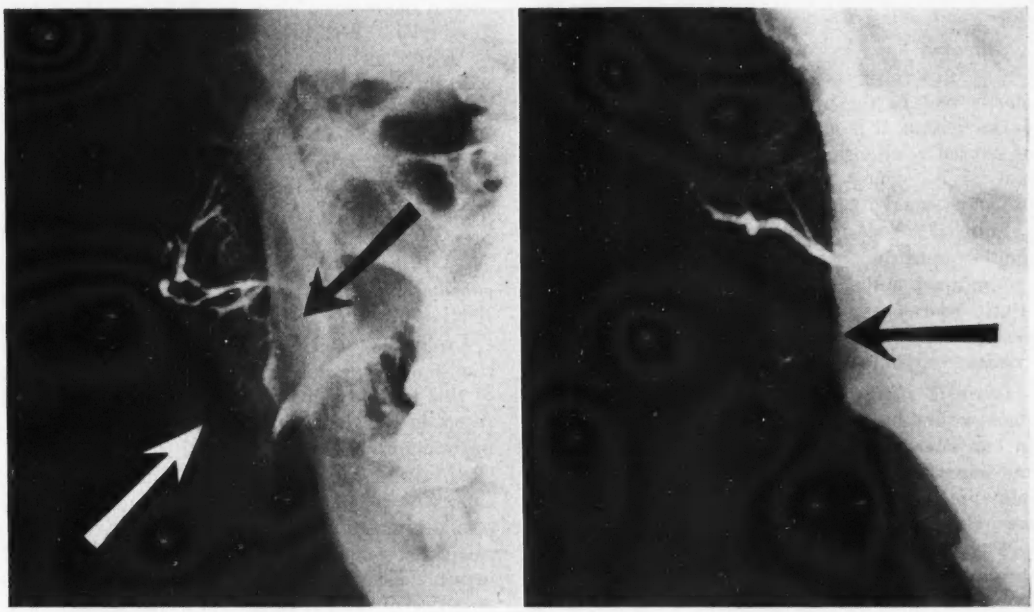


Figure 2.—Tumors in the pterygoid portion of the superficial lobe. The roentgenographic appearance of tumors in this location may be confused with that of deep lobe masses. *Left:* Indefinite mass of short duration. Infiltrating muco-epidermoid tumor. *Right:* Warthin's tumor, 2.5 cm. in diameter.

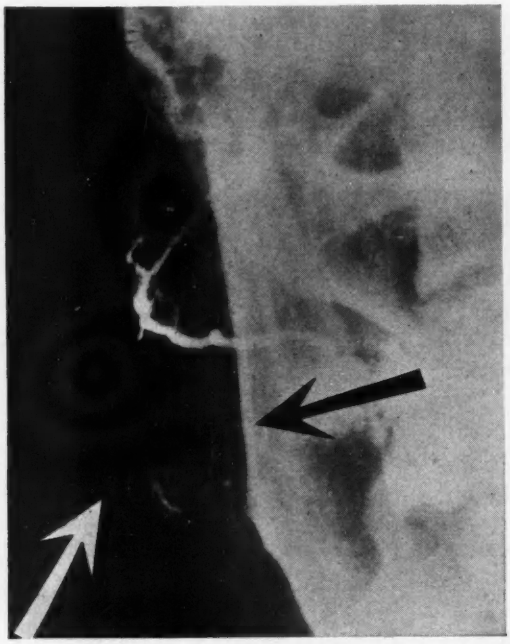


Figure 3.—Mass in the lower pole. Well-defined filling defect. Infiltrating muco-epidermoid carcinoma.

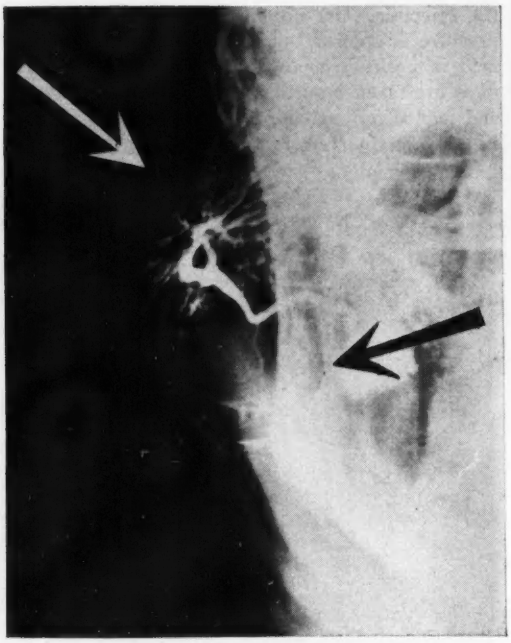


Figure 4.—Multiple Warthin's tumor, located in the pre-auricular portion, 1.5 cm. in diameter (white arrow), and in the deep lobe, 3 cm. in diameter (black arrow). The latter produces an "umbrella" effect, but may be confused with a pterygoid mass.

hours and can be overcome by massage and gum chewing. No further discomfort or complications were noted throughout the series.

Roentgenographic Interpretation

Although there are considerable anatomic variations in the size and shape of normal parotid glands and in the caliber and branching of the ductal system, it is not difficult to establish criteria of normal relationships. The normal appearance is that of a "winter tree" with evenly distributed branches gently tapering toward the periphery (Figure 1). A normal Stensen's duct is not more than 4 mm. in diameter.

Abnormal findings can be grouped as: (1) Intrinsic space-occupying lesions; (2) extrinsic indenting masses; (3) abnormalities within the ductal system.

Intrinsic space-occupying lesions are the most common findings and require anatomic localization. In our interpretation we distinguish between deep and superficial lobes, the latter being subdivided into pre-auricular, inferior pole, and pterygoid portions.

Well-defined masses may produce rounded pressure defects in the major and lesser ducts (Figures 2 and 3). Often the ducts are narrowed or stretched around the periphery of the mass, or the contrast medium does not fill the branches in the area of the mass. Deep lobe masses show a rather characteristic medial ballooning, which might be described as an "umbrella" effect (Figure 4). Masses compressing or displacing the distal part of Stensen's duct, as with tumors of the accessory lobes, are easily recognized (Figure 5).

Frequently, however, the mass cannot be clearly outlined. Then recourse must be taken to indirect evidence, such as ductal compression or displacement of a portion of the gland (Figure 2, *left*). Stereoscopy has been especially helpful in such cases. Previous operations in this region may cause considerable distortion of the normal anatomic relationships.

Extrinsic masses often cause indentation or rotation and displacement of the entire gland. Lesions of this type are due to enlarged lymph nodes adjacent to the gland, to tumors of the face and neck or to abnormal bone structures.

Abnormalities of the ductal system are usually caused by inflammatory (Figures 6 and 7) or neoplastic infiltrating processes (Figure 8). Central or peripheral calculi may be present. Occasional findings include variations in the caliber of the entire duct system, with irregular interruption of the continuity of the ducts, strictures and sacculations, the latter occurring especially at the terminal ends of

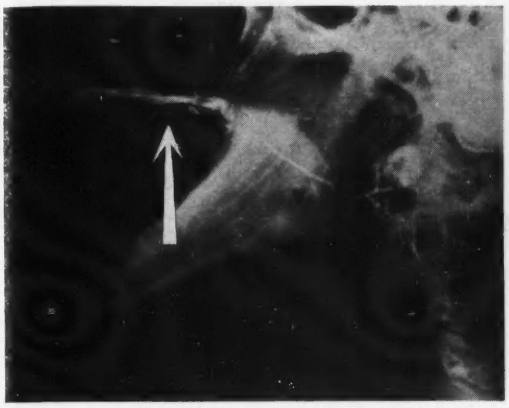


Figure 5.—Intrabuccal mass displacing the distal portion of Stensen's duct. Mixed tumor.

the ducts. Larger defects may form due to coalescence of smaller cavities. It is important to determine whether abnormalities of the ductal system are bilateral.

In the report on the roentgenological findings, location and size of the mass is described in detail. The possible nature of the mass is also noted—whether it might represent a solid tumor or a cyst; whether the changes suggest an inflammatory process or point to an infiltrating neoplasm; and finally whether there is suspicion of congenital sialangiectasis. Intravasation of opaque material would indicate cyst. Bilaterality in a patient of the younger age group would alert suspicion of congenital inflammatory process. Roentgen features typical of infiltrating neoplasm are shown in Figure 8.

Analysis of Roentgen Findings

In the 70 cases in which sialography was done, 36 space-occupying lesions were identified in the superficial lobe, 14 in the deep lobe, and three in accessory lobes. Two patients had diffusely infiltrating changes and nine had purely sialangiectatic changes. In six cases the findings were considered "normal." (In two of the "normal" cases there was slight displacement of the gland by what was interpreted as hypertrophy of the masseter muscle, and in one case there was generalized hypertrophy of the gland.)

Analysis of Surgical Findings

Fifty-eight patients were operated upon for total or subtotal removal of the gland. Included were all but one* of the patients' space-occupying masses; the two with diffuse infiltration of the gland; two patients with advanced sialangiectasis and sialadenitis. Two of the patients with normal x-ray findings but with palpable nodules were operated upon. One of them had had a mixed tumor removed previously. We found and removed two small recurrences. In the other case we found an enlarged lymph node, located superficially but within the substance of the gland.

*The patient, 79 years of age, had an easily palpable and visible mass, 2 cm. in diameter, in the pterygoid portion. According to the history the mass varied in size; one week later it had disappeared spontaneously, and was presumed to have been a cyst.

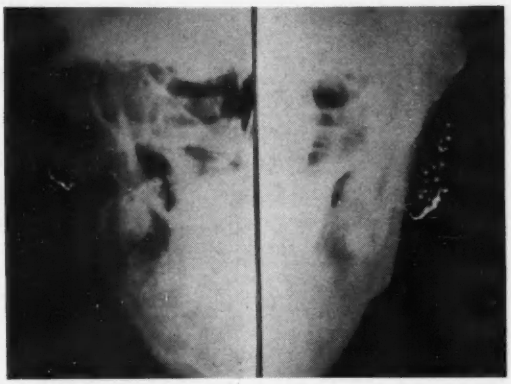


Figure 6.—Flat films demonstrate numerous small bilateral calculi. Bilateral congenital sialangiectasis. The patient had nontender intermittent bilateral swelling of the parotid glands, of long duration.

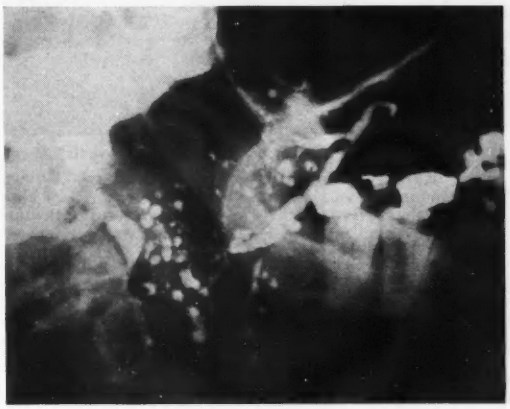


Figure 7.—Extensive sialangiectasis.

Correlation of Roentgen and Surgical Observations

The x-ray diagnosis proved correct in 50 of the 58 cases. In three cases in which the roentgenologic diagnosis was deep lobe masses, tumors in the pterygoid portion were observed at operation. The tumors were bulky and had an "umbrella" effect. In four other cases, roentgenographically the neoplasm was thought to involve the pterygoid and inferior portions but at operation was found to be in the deep lobe. These masses were all located posteriorly. In one case in which the roentgenogram was interpreted as normal, a small, superficial lymph

Figure 8.—Firm, diffuse tumefaction of the parotid gland, of several months' duration. Roentgenogram shows an ill-defined ductal system with variations of caliber and minimal sialangiectatic changes—typical of an infiltrating process. Subtotal removal revealed lymphoepithelioma of Godwin. Similar swelling of the opposite gland disappeared with moderate doses of roentgen therapy.

node within the substance of the gland was observed at operation.

The clinical impression—before sialography—was also correlated with the surgical findings. The clinical diagnosis was correct in 47 cases, incorrect in 11 cases. In all but one of the cases of incorrect diagnosis, tumors which were actually situated in the deep lobe had been assumed to be located in the superficial lobe.

DISCUSSION

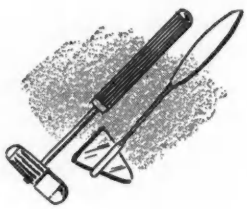
The present-day concept of operation upon the parotid gland calls for complete resection of the entire structure, or of one of the lobes, or of at least that portion of a lobe to which a mass is confined. The excision is performed without the surgeon's ever visualizing the mass. Presurgical localization of the tumor is of great help to the surgeon, as it may prevent injury to the facial nerve. This is of special importance in deep lobe masses. In the present series such localization was attempted by careful clinical and roentgenologic studies which were then correlated with findings at time of surgery.

Roentgenological interpretation was more often incorrect when the tumor was located in the pterygoid portion of the superficial lobe, thus producing the appearance of a defect in the deep lobe. Roentgenographic findings, however, were so frequently substantiated by surgical findings that in our opinion the use of sialography in the localization of parotid masses can be recommended. Even in the few cases in which the x-ray diagnosis was not completely in agreement with the surgical findings, the roentgenogram was not misleading to the surgeon.

Sialography is of particular value in the differentiation between surgical and nonsurgical diseases of the parotid gland, and often prevents needless surgical exploration.

The primary aim of many earlier reports in the literature was to determine whether sialography is of aid in arriving at a preoperative pathologic differentiation of parotid tumors. Most of these reports agree that roentgenograms permit recognition of inflammatory diseases, but they differ widely as to the value of x-ray findings for a preoperative pathologic diagnosis. It is our impression that roentgenograms do not permit a reliable pathologic diagnosis of parotid neoplasms.

300 South Beverly Drive, Beverly Hills.



Oral Administration of Orinase® (Tolbutamide)

Clinical Observations of Effect on Nondiabetic and Diabetic Humans

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SOLON PALMER, JR., *M.D.*, and LEE S. MONROE, *M.D.*, La Jolla

• **Ingestion of tolbutamide (Orinase®) by nondiabetic humans brought about a maximum reduction in blood sugar within one to two hours. In diabetic persons taking large doses of insulin, or who needed insulin for control, hyperglycemia, ketosis, and increased excretion of glucose in the urine developed when tolbutamide was substituted for insulin or was used before insulin therapy was begun.**

The only serious toxic manifestation observed was a skin rash in two patients.

Successful control of diabetes with tolbutamide was limited to cases in which the disease was of mild, stable type and the patient was 40 or more years of age, of normal weight, and with a previous insulin requirement of 5 to 30 units per day.

It was of benefit in 43.5 per cent of all diabetic patients in the series studied and in about 75 per cent of the group that might be referred to as selected. The duration of the disease and the duration of insulin therapy were unimportant in predicting effectiveness for tolbutamide therapy.

EFFECTIVE CONTROL of diabetes mellitus with an oral substitute for insulin has been the goal of investigators for many years. Early attempts as with the use of guanidin, a hepatoxin, were unsuccessful and it was not until 1942 that Janbon and associates[8,9] obtained the first encouraging results in the lowering of blood sugar with a sulfonamide. Loubatier's laboratory developed the subject and showed that in the absence of the pancreas this group of compounds was not effective. In 1955 Franke, Fuchs[7] and Bertram[1] and co-workers reported a sister compound (an aryl sulfonylurea called Carbutamide® or Bz55), and found it on clinical trial to be effective in lowering the blood sugar of elderly but not of juvenile diabetic patients. Shortly thereafter, a second aryl sulfonylurea, 1-butyl-3-p-tolsulfonylurea (Orinase® or tolbutamide), was brought forward and the available data on both compounds has been summarized.[5,17]

There is general agreement that the aryl sulfonylureas can reduce blood sugar in the majority of elderly persons with mild diabetes, but there is little or no effect on juvenile diabetes, ketosis, or in patients without a pancreas. It is felt that the mechanism of action is not like that of insulin, as glucose uptake is not obtained in eviscerated animals.[15,19] The most convincing theory to date is that the aryl sulfonylureas act by direct stimulation of the Beta cells of the pancreas although the action of the liver, insulinase,[13] thyroid[17] or glucagon[1] has been considered.

The potential hazards of prolonged administration of sulfonamides are considerable and include the gamut of hypersensitivities with the common cutaneous manifestations, leukopenia and full-blown collagen disease. Several deaths[12] have been attributed to the toxicity of Carbutamide (Bz55). On the other hand, tolbutamide (Orinase) has been associated with only minor toxic side-effects such as skin rash and leukopenia.

Although the convenience of administering an oral agent is considerable, this must be weighed against the toxicity of a product of relatively low therapeutic index.

From the Scripps Clinic and Research Foundation, La Jolla.
Submitted July 15, 1957.

The purpose of the present studies was to observe the effect of tolbutamide (Orinase) in persons with normal carbohydrate metabolism and patients with diabetes mellitus, taken at random. Patients requiring large doses of insulin for control were given a trial on tolbutamide in combination with reduced doses of insulin.

PROCEDURE

Eight nondiabetic patients were put in hospital for two days of study and observation before the administration of tolbutamide. The subjects remained at bedrest and on dietary regulation. Glucose tolerance tests were done before admittance, and each patient had a complete physical examination, blood cell count, urinalysis, cholesterol and urea determination and thymol turbidity test on the first day in hospital. Blood glucose determinations were made at 15 to 60-minute intervals for eight hours the second day, or the day preceding the oral administration of tolbutamide, on the day of tolbutamide administration and the day afterward. Blood for fasting blood sugar determination was taken at 8:00

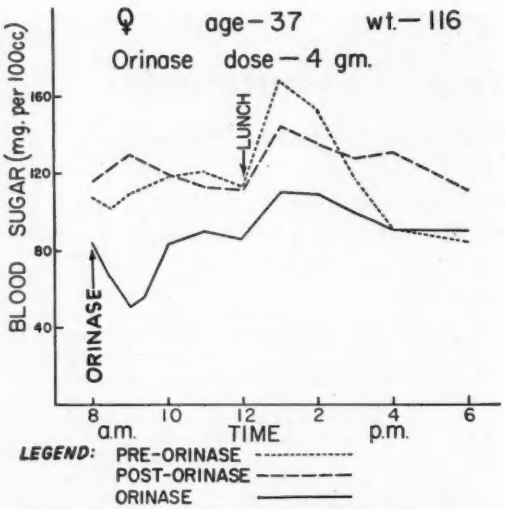

Chart 1.—Solid line demonstrates maximum response to single dose of 4 gm. of Orinase within first hour in normal 37-year-old woman. Broken lines represent blood sugar levels the day preceding and day following administration of Orinase. Subject did not have breakfast.

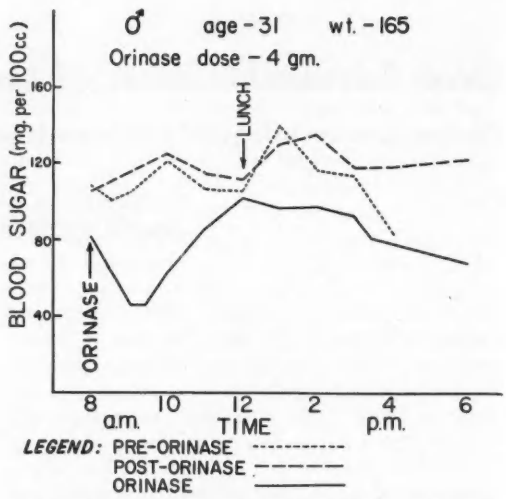

Chart 2.—Response to single dose of 4 gm. Orinase in a normal 31-year-old man is demonstrated by solid line. Note maximum blood sugar lowering effect within first hour. Subject did not have breakfast.

a.m. and Orinase then was given. The nondiabetic subjects were not given breakfast.

Thirty-seven diabetic patients, including 27 taking insulin (one of whom was juvenile), were kept in hospital for from two to four weeks and an attempt was made to control the diabetes with tolbutamide alone or in combination with insulin. Before therapy, these patients had the following studies: Pulmonary roentgenogram, electrocardiogram, complete blood cell count, determinations of protein-bound iodine (PBI), iodine^{131} uptake and blood urea, urinalysis, and phenolsulphthalein, bromsulphalein and thymol turbidity tests. After therapy was begun, examinations of the urine sediment and tests for urinary albumin, as well as blood cell counts were carried out every other day. Each week a phenolsulphthalein test and blood urea determination were done, and at two-week intervals an electrocardiogram was made and bromsulphalein, thymol turbidity, protein-bound iodine and I^{131} determinations were performed. Results of these tests were within normal limits. In addition to the foregoing tests, urinary glucose excretions for 24 hours were determined. In the diabetic patients blood for fasting blood sugar determination was taken at 7:30 in the morning and Orinase was given immediately afterward. They ate breakfast at 8:00 a.m.

RESULTS IN NONDIABETIC PERSONS

Charts 1 and 2 show the effect in a normal 37-year-old woman and a normal 31-year-old man. In those patients the maximum response was within

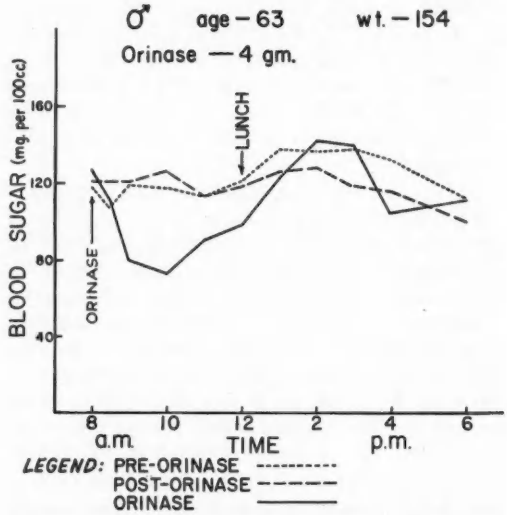

Chart 3.—Maximum effect of single dose of 4 gm. of Orinase within first two hours in 63-year-old nondiabetic man. Subject did not have breakfast.

the first hour. It was within the first two hours in a 63-year-old man, Chart 3. A comparison between the effect of Orinase and Orinase sodium was observed in a woman 71 years of age, Chart 4. The major difference between these two compounds was in their solubility characteristics, the sodium salt being extremely water-soluble and the free sulfonamide form being highly insoluble. We were interested in comparing the relative activity of the two tablet formulations as far as acute blood sugar lower-

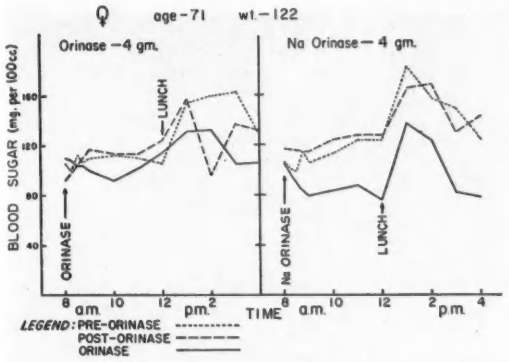

Chart 4.—Comparison of response to single doses of Orinase and sodium Orinase in 71-year-old nondiabetic woman. Subject did not have breakfast.

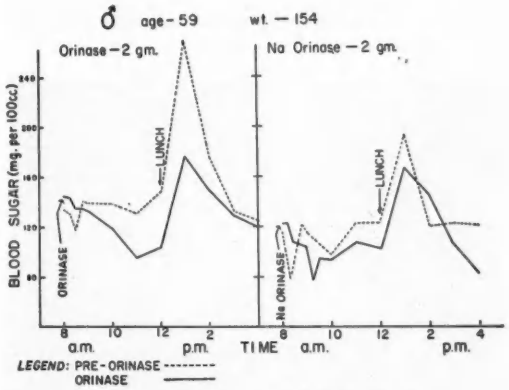
♂ age-59 wt. - 154
Orinase - 2 gm.
Na Orinase - 2 gm.
BLOOD SUGAR (mg. per 100cc)
LUNCH
ORINASE
Na ORINASE
8 a.m. 10 12 p.m. 2 TIME 8 a.m. 10 12 2 p.m. 4
LEGEND: PRE-ORINASE
ORINASE

Chart 5.—Comparison of response to single doses of Orinase and sodium Orinase in a 59-year-old nondiabetic man. Subject did not have breakfast.

ing effect was concerned after oral administration. The 71-year-old woman mentioned had a delayed response in the afternoon after Orinase and the response after sodium Orinase was continued through the day.

A comparison of effect of the two drugs in a 59-year-old man is shown in Chart 5. He was one of the first patients in the investigation and at that time the dose of Orinase given to nondiabetic persons for experimental purposes was 2 gm. Later we discontinued the use of sodium Orinase because we could not see that it was more advantageous than Orinase.

In Chart 6 a comparison is made of the response in a nondiabetic woman and a diabetic woman after a single dose of 4 gm. of tolbutamide. There was a sudden decrease in the blood sugar level of the nondiabetic patient within the first hour, and a gradual decrease in the blood sugar level of the diabetic patient over five hours. Both women were 59 years of age.

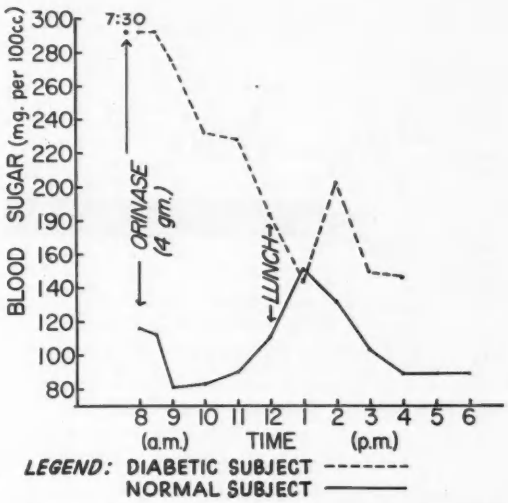

Chart 6.—Comparison of response to single dose of 4 gm. of Orinase in a 59-year-old diabetic woman and 59-year-old nondiabetic woman. Note gradual decrease of blood sugar level over five hours in the diabetic woman and sudden decrease within first hour in the nondiabetic one. Neither of these subjects had breakfast.

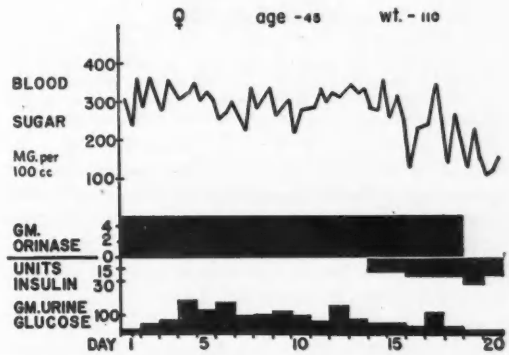

Chart 7.—Blood sugar levels and 24 hours urinary glucose output of 45-year-old woman with newly discovered diabetes during administration of Orinase for two weeks and combination of Orinase and insulin for six days.

RESULTS IN DIABETIC PERSONS

Three types of diabetic patients did not respond to Orinase. Chart 7 shows the effect in the case of a thin, 45-year-old woman recently found to have diabetes. She had never taken insulin. Her blood sugar levels and 24-hour glucose output during the time of treatment with tolbutamide are shown. Chart 8 shows the blood sugar levels of a 68-year-old white man, who had been taking 30 units of NPH insulin daily. He did not respond satisfactorily to Orinase and use of the drug was discontinued. Chart 9 shows the effect on an 8-year-old boy with newly discovered diabetes who had never received insulin. During the

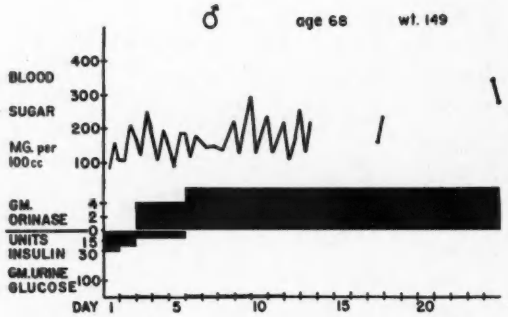

Chart 8.—Blood sugar levels and urinary glucose output of 68-year-old diabetic man who did not respond satisfactorily to Orinase. After hospitalization for two weeks he continued under observation as an outpatient.

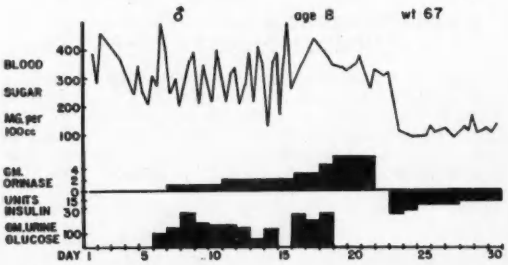

Chart 9.—Effect of Orinase on an 8-year-old boy with newly discovered diabetes. He had never taken insulin. Note the decrease in blood sugar level the first day insulin was administered after Orinase was discontinued.

last day of tolbutamide therapy the boy received up to 6.5 gm. without appreciable effect. Diabetes was eventually controlled by administration of 20 units of insulin daily, but the dramatic initial fall in blood sugar occurred following administration of 40 units for the first and 35 units for the second day.

As was stated previously, no abnormality was noted in results of the many tests made for evidence of toxicity. Crystalluria, hematuria, leukopenia and the commoner effects of sulfonamide therapy were not observed in any of the patients.

When the urine was acidified with a strong acid, a precipitate was observed. This was the oxidized form of the drug and under certain circumstances might be mistaken for albumin. However, 5 per cent acetic acid was not sufficiently strong to cause this precipitate, therefore the use of heat and acetic acid was a reliable test for albumin in the patients taking this drug.

Dermatitis developed in two patients during treatment—an erysipeloid eruption on the face in one case and a fairly generalized morbilliform eruption in the other. The erysipeloid lesion developed after two months of taking 4 gm. of Orinase daily. The eruption cleared completely within seven days after discontinuance of Orinase. Then a provocative dose

Figure 1.—Positive reaction to skin tests with 10 mg. of sodium Orinase on the arm of a patient who had had erysipeloid eruptions on face after two months of taking Orinase, then prompt clearing of lesions after discontinuance of the drug.

Figure 2.—Morbilliform eruption on 60-year-old diabetic man who had been taking Orinase for 14 days (4 gm. daily the first week and 2 gm. daily the second week).

of the drug was given and an exact duplication of the former lesions appeared.

Figure 1 shows positive reactions to skin tests with 10 mg. and with 100 mg. of sodium Orinase. The positive reaction at the site of intracutaneous injection of 100 milligrams of sodium Orinase was suspected to be due in part to chemical irritation, and that amount of the drug is not reliable for ascertaining hypersensitivity. The antibodies that were present in the patient with erysipeloid dermatitis were passively transferred to normal recipients. Skin tests with 10 mg. of sodium Orinase in 15 normal controls, and six normal controls receiving Orinase, gave negative reactions.

Figure 2 shows a morbilliform eruption. Hypersensitivity could not be demonstrated by skin tests with sodium Orinase.

One patient with successful control of diabetes objected to taking tablets and stopped the drug.

In the group of 17 patients requiring insulin for the first time or returning to insulin for control, not one was overweight or was considered to have mild diabetes. The age range was from 8 to 69 years.

In the patients requiring large doses of insulin, hyperglycemia, ketosis and increased excretion of glucose in the urine occurred when an attempt was made to substitute Orinase for insulin.

Table 1 summarizes the essential data on the 16 successfully treated patients. All were 44 years of age or older; the range was from 44 to 70 years, and the average age 59 years. No patient who required large doses of insulin could be successfully managed with tolbutamide. Daily insulin requirements of patients with successful control by tolbutamide varied from 5 units to 30 units and the mean was 16 units. In patients who were not managed adequately with tolbutamide, control was reestablished on the same dietary regimen and insulin dosage as had been employed before the experimental period.

Urinary excretion of sugar was measured before and during tolbutamide therapy. When diabetes was well controlled by insulin, the average daily excretion of sugar was 2.6 gm. with ranges from 1 to 9 gm. With tolbutamide therapy the average daily excretion was 1.8 gm. and the range was 0.3 to 6.4 gm. Although these differences are small, they probably reflect the longer and more evenly sustained hypoglycemic action of tolbutamide. Dosages of tolbutamide varied. At first each patient was given 3 gm. daily in divided doses. Adjustments were made periodically thereafter in accordance with the individual needs as indicated by repeated determination of the content of sugar in the blood and urine. Doses as high as 6 gm. a day were used, but in general when results could not be obtained with 4 gm. daily, larger amounts were of no avail.

TABLE 1.—*Data on Group of 16 Patients Treated Successfully with Orinase*

	Average	Range
Age	59 yrs.	44-70 yrs.
Insulin units required daily	16	5-30
Orinase dosage (gm./day)	3	0.5-6
24-hour urinary glucose (gm.):		
On insulin	2.6	1-9
On Orinase	1.8	0.3-6.4

COMMENT

Of the 37 diabetic patients taken at random who were treated with tolbutamide (Orinase), control of diabetes was considered adequate in 16 (43.5 per cent). This is a somewhat lower proportion than the 81 per cent[16] and 73 per cent[2] reported by other investigators, but it compares relatively well with the 48 per cent reported by Camerini-Davalos, Marble and Root[3] in a series of cases selected to include only diabetes of the adult or stable type. Similarly, in another series 80 per cent of more than five hundred ambulatory adult diabetic patients had adequate control with tolbutamide alone.[6] In the present series, control was satisfactory in 16 of the 21 patients who were considered most likely to respond to treatment wih tolbutamide. Criteria for success in this series were as follows:

1. Maintenance of reasonable blood sugar levels (less than 180 mg. per cent three hours postprandial)
2. Reasonable 24-hour urinary excretion of sugar (less than 7.0 gm.)
3. Prevention of ketosis
4. Absence of symptomatic hypoglycemia
5. Tolerance of drug without toxicity.

As was mentioned in a recent editorial,[4] there are many persons with mild diabetes who border on requiring insulin, or who refuse to take insulin for moderate hyperglycemia. More exact control may be possible with tolbutamide, and in the absence of toxicity this advantage should probably be utilized until relationships between inadequate control and severity of complications are more precisely defined. In the present series no patient, whether successfully treated or not, responded better to a combination of tolbutamide and reduced doses of insulin than to tolbutamide alone. All patients responding well to tolbutamide in this series were surveyed in an effort to unveil any possible common denominators. In general, the following factors applied uniquely to the group in which control was obtained with tolbutamide:

1. The patients were more than 40 years of age.
2. The disease was of the milder, more stable type.
3. Insulin requirements were minimal to moderate.

There was no specific correlation between duration of diabetes and probability of favorable response to tolbutamide. Of much greater importance was the character of the diabetes present. All who responded well had adult, stable diabetes. Hypoglycemic reactions often occurred in normal volunteers who were given tolbutamide, but such reactions were quite rare in patients having diabetes.

Frequent follow-up studies on this original group and other successfully treated patients for a period of 14 months indicated that there is a tendency for the blood sugar level to decline gradually in many cases. Consequently, we have been able to reduce the dosage of tolbutamide to 0.5 gm. daily in such patients.

ACKNOWLEDGMENTS

Grateful acknowledgment is made to Dr. C. J. O'Donovan of the Upjohn Company for supplies of tolbutamide (Orinase); to Dr. Wayne Monsees, ophthalmologist, for his careful observations of the patients investigated; and to Mrs. Barbara Reeves and Mrs. Anna McEuen for their technical assistance in this study.

ADDENDUM

Follow-up studies recently showed a significant decrease in I^{131} uptake and protein-bound iodine in one patient, a 68-year-old woman, who had been taking Orinase for about ten months. The protein-bound iodine, was 6 mcgm. per 100 cc. and I^{131} uptake 11 per cent on May 1, 1956. On February 23, 1957, the protein-bound iodine was 2.9 mcgm. per 100 cc. and I^{131} uptake on February 26, 1957, was 5 per cent. Orinase was discontinued on February 27, 1957, and two days later on March 1, 1957, the I^{131} uptake was 22 per cent. Physical examination and interval history gave no evidence of hypothyroidism.

The beneficial effect of tolbutamide seems to have become exhausted in another 44-year-old woman who had been receiving the drug for ten months. Blood sugar levels three hours postprandial are now above 180 mg. per 100 cc., and it has been necessary for her to resume use of 20 units of NPH insulin daily for proper control of diabetes.

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Prosthetic Arteries

Resection of the Abdominal Aorta and Replacement with Plastic Materials

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• In seven cases, nine operations were done in which arterial replacements made of synthetic fabrics were used. In three cases the operation was entirely elective, and at last report the patients were doing well. In the four other cases there were acutely complicating factors, known before operation, and the patients died although so far as functioning of the prosthetic artery was concerned there was difficulty in only one case—a leak at one of the anastomotic seams. In one case in which the synthetic material was removed after it had been in place some time, the prosthesis showed little effect of foreign body reaction.

DIRECT SURGICAL operation is now an accepted treatment for aneurysmal and obliterative diseases of the abdominal aorta.

Abdominal aortic aneurysm frequently results in death from rupture of the aneurysm. Persons with abdominal aortic aneurysms have a lowered life expectancy.[4] Without surgical intervention, following perforation of an abdominal aortic aneurysm, the disease is nearly always fatal. Cooley and DeBakey,[1] Gerbode,[5] Julian[6] and Shumacker[7] have all shown that early surgical intervention can convert a hopeless prognosis into a relatively more favorable one. Wylie[9] said that whereas the mortality rate for resection and grafting of aneurysms of the abdominal aorta is about 10 per cent if operation is done before rupture of the vessel occurs, it is 50 per cent if delayed until after rupture. In view of the high incidence of eventual rupture, in most instances verification of the existence of an aortic aneurysm is an indication for operation even in the absence of pain.

Most reports of resectional operation on the abdominal aorta indicate that the replacement has nearly always been by use of a homograft. Deterling[2,3] reported a great deal of investigative work on evaluating synthetic materials and fabrics suitable for blood vessel replacement. He said that, in peripheral arteries, homografts appear to be superior in patients with segmental occlusive disease. Szilagyi[8] also expressed belief that homografts are still the best arterial substitutes.

It would appear that the most satisfactory use of a synthetic prosthesis would be as a substitute for a segment of a large caliber vessel in a relatively immobile area—the abdominal aorta, for example.

The following cases represent patients in whom synthetic prostheses have been used. The types used were either the Orlon prosthesis developed under the direction of Dr. Paul Sanger, or the Edwards-Tapp seamless prosthesis. In the one case requiring multiple operations, both were used.

CASE 1. The patient, a white man 40 years of age, was admitted to the hospital June 27, 1956, with a diagnosis of obliterative arteriosclerosis of the terminal aorta. For two years he had had severe cramping pains in both thighs, hips and legs and had been unable to walk far without acute pain. Upon examination, the blood pressure was 130/77 mm. of mercury. The heart was not enlarged. All arterial pulses in both lower extremities were absent.

At operation, advanced atherosclerosis of the terminal aorta and the common iliac arteries was noted. Resection of these vessels was carried out, the proximal extent being just distal to the renal arteries. Retrograde flow was obtained bilaterally, but was less forceful on the left side. A Sanger prosthesis was used, and lumbar sympathectomy was done on the left side. After operation satisfactory arterial pulses were present at all times on the right but not on the left. The patient was discharged, ambulatory, on the fifteenth postoperative day.

The patient continued to have pain in the left lower extremity when walking, but was essentially free of pain in the right leg. It was believed that further operation could give additional relief.

Operation was done in January, 1957, to explore the arterial supply to the left lower extremity and, if possible, to provide improvement. On the left, the artery was found to be obliterated just distal to the prosthesis. The prosthesis substituting for the left common iliac artery was compressed and contained no blood up to the bifurcation. It was possible to open this limb and obtain normal arterial flow through it. An Edwards-Tapp arterial substitute was then sutured to the distal end of the reopened prosthesis proximally, and was anastomosed end-to-side with the left common femoral artery. Pulses in the left lower extremity then were excel-

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lent. On the fifth postoperative day these pulses suddenly disappeared.

At a third operation a large embolus was found in the left limb of the prosthesis. For fear of compromising the situation on the right (the good side) by attempting complete removal and replacement of the left limb alone, it was decided to resect all the previous prostheses except for the very uppermost portion of the aortic limb. An Edwards-Tapp replacement was then used and extended distally to be anastomosed end-to-side with the common femoral arteries. Good pulses were present bilaterally at the conclusion of the operation. In the immediate postoperative period thrombophlebitis involving the deep venous system of the left lower extremity developed. The patient was discharged from the hospital March 7, 1957, ambulatory and with good arterial pulses in both feet. When examined at intervals later, he had good pulses in both lower extremities, was working, and was remarkably free of symptoms.

The excised prosthesis which had been in use for about seven months was examined microscopically and showed only mild foreign body reaction.

CASE 2. A 73-year-old white man, admitted to hospital September 12, 1956, had a history of pain in the back and mid-abdomen for three months. The blood pressure was 200/100 mm. of mercury. A large pulsating mass was felt in the abdomen. No abnormal calcification or mass was demonstrated by anteroposterior and lateral x-ray views of the abdomen.

At operation a large arteriosclerotic aneurysm of the abdominal aorta was resected. A Sanger prosthesis was used, extending from just distal to the renal arteries to just proximal to the termination of the common iliac arteries. A good retrograde flow was obtained from both iliac arteries. The postoperative course was uneventful and the patient was discharged, ambulatory, on the tenth day. He was examined from time to time afterward, and good arterial circulation to the lower extremities always was present.

CASE 3. A white man 65 years of age was admitted to hospital February 15, 1957, with a history of pain for two months in the lumbar area and radiating into the inguinal regions and thighs. The blood pressure was 160/90 mm. of mercury. Pulses were present in both lower extremities. Radiographs showed dilatation of the lower abdominal aorta with calcification, and dilatation of the common iliac arteries with much calcification. No abnormalities were noted in an electrocardiogram. The nonprotein nitrogen content was within normal limits.

At operation an arteriosclerotic aneurysm involving the distal abdominal aorta, the common iliac arteries and the hypogastric arteries was observed. The external iliac arteries were pliable to palpation and in very decided contrast to the involved vessels. The aneurysm was resected, the hypogastric arteries were ligated and a Sanger prosthesis was placed between the aorta and the external iliac arteries. The patient left the hospital ambulatory and with good arterial pulses in both lower extremities on the sixteenth postoperative day. He continued to do well.

CASE 4. The patient, a white man 66 years of age, had been under observation as an outpatient for about one year and was known to have an aneurysm of the abdominal aorta, bronchiectasis, pulmonary emphysema and generalized arteriosclerosis. He was admitted to hospital December 26, 1956. The risk of operation in his particular case had been explained to him previously and he had decided against it. On admission, however, he was having much discomfort in the lower extremities, and for several weeks had noted swelling of both lower extremities. It was felt that the swelling was due to vena caval obstruction. The blood pressure was 124/72 mm. of mercury. The patient at this time was eager for surgical treatment in spite of the risk.

At operation, January 2, 1957, a large arteriosclerotic aneurysm of the abdominal aorta with inflammatory reaction involving the common iliac veins and the distal vena cava in a dense compressive mass was observed. The aneurysm was resected and a Sanger replacement used. The patient tolerated the procedure remarkably well and had good pulsations in the lower extremities when it was completed. The swelling in the lower extremities disappeared quickly. On the tenth postoperative day, evisceration of the abdominal wound occurred. Reclosure was carried out and the patient did fairly well for several days. He died on the twenty-fourth postoperative day, however, of bronchopneumonia, pulmonary infarct and jejunal obstruction due to adhesions.

CASE 5. A white man 58 years of age was admitted to the hospital in February, 1957, after having collapsed in a physician's office. He early complained of much abdominal pain and the signs of severe shock were present. A diagnosis of ruptured aortic aneurysm had been made. At the time we were consulted the patient had been in shock for seventeen hours. It appeared that bleeding had stopped and that the most urgent problem was of blood replacement and of determining urinary function. The patient responded to transfusion and there was good urinary output in the next 24 hours. The blood pressure gradually rose to 200 mm. of mercury systolic. The patient suddenly had severe pain again and was suddenly in shock. It was felt that the only hope lay in immediate operation. A ruptured abdominal aortic aneurysm was resected and a Sanger prosthesis was used. At the conclusion of the operation the blood pressure was 120 mm. of mercury systolic and the general condition of the patient was fair. Pulses were present in both lower extremities. The patient's condition, however, did not improve, and he died seven hours after operation. At autopsy, unusually advanced and extensive

generalized arteriosclerosis, pronounced coronary artery atherosclerosis and coronary occlusion with early infarct were noted. There was a leak at the upper junction of the prosthesis and the aorta. The pathologist reported that even if the patient had survived the immediate acute problems, his life expectancy could not have been more than a few months.

CASE 6. The patient, a 71-year-old white man, was first observed in the hospital in March, 1957, with a diagnosis of intra-abdominal hemorrhage due to ruptured aneurysm of the abdominal aorta. The patient was moribund, but both he and his family wished even the slight chance that operation offered. At operation a large ruptured aneurysm was found and resected. An Edwards-Tapp prosthesis was used from the level of the renal arteries to the common femoral arteries. It was necessary to clamp above the renal arteries for 33 minutes. At the conclusion the patient's immediate condition was somewhat better than preoperatively. Then, a urinary shut-down developed and the patient died the following day.

CASE 7. A white man 58 years of age entered the hospital December 5, 1956, with a history of pain in the lower abdomen and back for four months. The pain improved for a time, then became worse a month before admittance to hospital. About two weeks previously a mass had been noted in the right lower quadrant of the abdomen. Upon examination the mass was observed to be nontender and to have no noticeable pulsation. The blood pressure was 186/120 mm. of mercury. In an x-ray film of the chest, redundancy and dilatation of the aorta, particularly the ascending portion of the arch, were noted. At operation an aneurysm of the common iliac arteries and the terminal aorta was observed. Dissection had occurred and a massive hematoma occupied the retroperitoneal space of both lower quadrants of the abdomen and of the entire posterior one-third of the pelvis. The old blood was evacuated and the aneurysm resected. A Sanger prosthesis was used and it functioned well. The postoperative course was uneventful and the patient was discharged, ambulatory and with good pulses in both lower extremities, on the eleventh postoperative day. He returned to work and did well until about six months later, when he was returned to the hospital by ambulance. Shortly after admission he died of massive hemoptysis due to rupture of an intrathoracic aortic aneurysm. At postmortem examination the prosthesis was observed to be intact and functionable.

TECHNIQUE

In the operation used in the cases here reported, the anastomosis is made the same as in using a homograft. This consists of one row of continuous 4-0 arterial silk with an occasional interrupted reinforcing suture. It is important that the lumen of the replacement not be allowed to fill with blood during the period of anastomosis, as clotting will occur and may cause obstruction at some distal area in the arterial tree. There are at least two other technical points of importance. We feel that with the materials that are available at present, the prostheses should be used only as replacements for large arteries located anatomically in a relatively immobile area. In replacing the terminal aorta there is an advantage to be gained by shortening the aortic segment and proportionately lengthening the iliac segments, thereby reducing the angulation of the iliacs at the bifurcation.

COMMENT

In only three of the seven cases presented could operation be called purely elective. The three patients continue to do well. In three of the remaining cases the grave complication of dissection and hemorrhage were factors. In the other case the patient had known pulmonary disease plus the complication of vena caval obstruction.

As yet, insufficient time has elapsed to determine the long range results of the use of prosthetics as large artery replacement material in human patients. Undoubtedly, the ideal material has not yet been produced, although the improvement has been rapid.

There are several advantages of the synthetic replacements. Storage is not a problem and they are readily available. They are easier and faster to use from a technical standpoint. There is reason to believe that extensive tortuosity or dilatation will not develop in them. Certainly they enlarge the geographical horizons for saving life and limb in times of emergency.

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Malignant Carcinoid Tumor

Altered Reactivity of the Skin to Serotonin in Two Patients

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• **Appropriately controlled tests were carried out in two cases of patients with carcinoid tumors and abnormal circulating levels of serotonin to verify a hypothesis that such patients would have a greater tolerance for exogenous serotonin than would normal persons. Contrary to the hypothesis, the patients had not less but more reaction to intradermal injection of serotonin than to histamine and also more reaction than normal persons.**

If this altered reactivity were to be observed consistently in large series of patients with malignant carcinoid disease, it might be used as a presumptive test.

IT HAS BEEN SUGGESTED that serotonin may be a naturally occurring vasodilator compound important in vascular homeostasis because of its ability to regulate peripheral neurogenic vasomotor tone.[4]

The syndrome of a carcinoid tumor with metastasis is now well recognized.[3] Unfortunately the many real and surmised biological functions of serotonin are not well understood.[4]

It was conjectured that a patient with a carcinoid tumor and abnormal circulating levels of serotonin would be able to tolerate exogenous serotonin to a greater degree than a normal person, and that if so the skin reactivity to serotonin as compared with reactivity to histamine would demonstrate the phenomenon. Tests in point were carried out in two cases.

REPORTS OF CASES

CASE 1. A white man 56 years of age, had flushing and peripheral cyanosis, episodic bouts of diarrhea and a large nodular liver. He had had no asthmatic-like bouts. No cardiac murmur could be heard. He received no medications in the week preceding skin testing. In roentgen studies of the upper gastrointestinal tract, pronounced hepatomegaly, with displacement of the stomach laterally and posteriorly, was noted. Excretion of 5-hydroxy-indole acetic acid in four 24-hour periods was as follows: July 17, 38 mg.; July 4, 52 mg.; June 28, 46 mg.; June 24, 45 mg. Biopsy of the liver at laparotomy confirmed the diagnosis.

CASE 2. A Negro woman 56 years of age had a grade II systolic murmur heard along the left sternal border, possible beginning bouts of diarrhea, and hepatomegaly. She had had no asthmatic-like bouts, no episodes of flushing or cyanosis, no symptoms of gastric ulcer. The patient was receiving tetracycline for a urinary tract infection. Excretion of 5-hydroxy-indole acetic acid for two 24-hour periods was as follows: September 9, 129 mg.; August 21, 118 mg. Biopsy of a yellow nodule on the liver through a peritineoscope was diagnostic of malignant carcinoid growth.

The materials used were: tuberculin syringes with No. 25 (French) needles; serotonin creatinine sulfate in physiological saline solution in dilutions of 1:1000 and 1:2000; histamine phosphate solution in physiological saline solution in dilution of 1:4000; and physiological saline solution.

Method: In each case two doses, one of .05 ml. of 1:1000 serotonin and one of 1:2000 serotonin, were administered intradermally in the flexor surface of the right forearm in a proximal and distal position, respectively. Histamine 1:4000 and a physiological saline solution control were placed on the left forearm in a similar fashion.

Results

CASE 1, right arm: The flexor surface of the forearm became diffusely erythematous and the finger tips became cyanotic. This occurred in slow progression and was complete in seven minutes. Then erythema slowly disappeared and the skin appeared completely normal in 25 minutes. No whealing was noted. The patient noticed pruritus without prompting. No systemic manifestations were noted. An hour later the same procedure on the other arm evoked the same response.

Left arm: At the site of histamine injection there was an area of erythema 3 cm. in diameter and a wheal 1.25 cm. in diameter, without pseudopod formation. The patient spontaneously complained of pruritus in this area. No reaction occurred at the site of saline solution injection. The histamine reac-

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tion resolved completely in two hours and fifteen minutes.

CASE 2, right arm: The two test areas become confluent in a patch of erythema 8 cm. by 10 cm. This reaction occurred quite rapidly, was complete in three minutes and had completely disappeared in seven minutes. No whealing was noted. Even with questioning, no subjective complaints were made regarding pain or pruritus. There was no fingertip cyanosis or manifestation of systemic effect.

Left arm: At the site of histamine injection an area of erythema 2.25 cm. in diameter and a wheal 2 cm. in diameter developed. One long trailing pseudopod was noted. The patient made no sensory complaint. No reaction was noted at the site of saline solution injection.

Twenty-five persons without disease were tested in a similar manner and the area erythema at the histamine injection was comparable in size to the area at the serotonin injection site. No whealing was noted in the area of intradermal serotonin in any of the controls. Eleven of the controls noted pruritus and pain to histamine and serotonin respectively. Nine reported no sensation of itch or pain. Two noted pain to both. One noted pruritus to both. One noted pain to histamine and pruritus to serotonin. One noted pruritus to histamine and no sensation to serotonin.[1,2]

Discussion

Contrary to the hypothesis, the two patients tested were not able to tolerate intradermal serotonin as well as normal persons. In supposition, one could postulate that patients with malignant carcinoid growths hyper-react to intradermal serotonin because their capillary threshold is altered by the exogenous increase of serotonin, more so than the normal.

If this altered reactivity of the skin is demonstrable in more than two patients with malignant carcinoid disease, a possible presumptive skin test is prognosticated.

Conclusion

Two patients with abnormal concentrations of 5-hydroxy-indole acetic acid in their urine had hyper-reactivity to intradermal injections of serotonin.

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Social Security Footnotes

OLD AGE AND SURVIVORS INSURANCE is a system under which the active workers and their employers are contributing the taxes necessary to pay benefits to their fellow citizens on the benefit rolls. The active workers now covered under the system must look for their own old-age benefits, not in any large measure to the Trust Fund, which is only a moderate buffer fund to cover temporary excess of benefit payments over tax receipts, but mainly to the willingness of the next generation of active workers to pay the increased taxes out of which the retirement benefits will come.

—From the Department of Public Relations, American Medical Association

Stapes Mobilization for Otosclerotic Deafness

The Monitored Peribasal Technique

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• **Direct surgical operation on the stapes footplate region in the treatment of hearing loss due to otosclerosis has been revived.**

In the last 100 cases of a total series of 600, the peribasal stapes mobilization (stapedolysis) technique was completely successful in 62 per cent, partially successful in 29 per cent and a failure in 9 per cent.

Stapes mobilization requires precise microsurgical technique monitored by audiometry during the surgical procedure.

It appears that mobilization is the preferred approach to the surgical treatment of otosclerosis, but fenestration of the vestibular labyrinth is an important secondary procedure in some cases.

Successful restoration of hearing requires adequate mobility of the footplate coupled with physiological continuity of the drum ossicular chain mechanism.

"CLINICAL OTOSCLEROSIS" is the most common cause of deafness in adults. Histologically, the lesion is bony dystrophy originating in the labyrinthine capsule. Starting as "otospongiosis" it matures into "otosclerosis." In its most common form, the lesion invades the anterior limb of the stapes footplate and the medial aspect of the anterior stapedial crus. This bony bridge produces a mechanical obstruction to normal transmission of acoustic energy to the vestibular perilymph. The impedance matching effect of the middle ear mechanism is thus lost and "conductive deafness" ensues. The basic cause of otosclerosis remains obscure, and there is no known method of prevention or of medical therapy.

The symptoms of otosclerosis are deafness, tinnitus, and occasionally vertigo. The onset is insidious, usually bilateral, and slow in rate of progression. It is more common in females. It starts in the second or third decade of life and usually reaches a stable plateau in the fourth or fifth decade. Usually no abnormalities can be seen otoscopically. The canal, drum, middle ear and eustachian tube are normal. Results of tuning fork tests and audiometric examination are typical of conduction deafness. The result of the Rinne test is negative, bone conduction (BC) of sound being greater than air conduction (AC), and there is a significant bone-air gap* on pure tone audiometry. Vertigo occurs in occasional cases but is not usually severe; it may resemble that of Meniere's disease.

The surgical approach to this problem had its origin in 1876 with the observation by Kessel[7] that manipulation of the stapes could improve the hearing in stapes ankylosis. Further surgical intervention by Miot[9] in 1892 showed that this idea was practical. However, for various reasons this surgical method was temporarily dropped. It was not until several decades later that an indirect or detour surgical route through the vestibular labyrinth was devised, and it was finally perfected by Lempert[8] in 1938.

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*The bone-air gap is the expression in decibels of the degree of conductive deafness.

In 1952, Rosen[10] revived interest in the direct surgical attack on the stapes itself. Since then the direct operation has been shown effective in a large number, perhaps a majority, of patients with otosclerotic deafness.

In view of experiences of the past few years, stapes mobilization, the direct operation, appears to be the preferred primary surgical treatment for otosclerotic deafness. Fenestration of the horizontal semi-circular canal now takes a secondary but very important position in the surgical plan. The otosclerotic patient deserves a total approach to the problem of surgical management. Ideally, the surgeon who undertakes the stapes mobilization procedure should be prepared by training and experience to subsequently perform a fenestration operation if necessary.

As has been pointed out in previous publications, physiologically the stapes mobilization (stapedolysis) approach is preferable. It is a direct operation upon the mechanical lesion and does not destroy the efficacy of the normal middle ear mechanism. It not only is of great value in patients with moderate losses who can hope for restoration of normal hearing, but is of great value in patients with far advanced disease with secondary cochlear involvement who cannot hope for normal hearing but may hope

for a significant improvement which will permit better utilization of amplification through a hearing aid. The operation can be done on patients of almost any age, and the morbidity factor is very low. It is, however, a precise and highly intricate anatomical procedure which requires dextrous micro-surgical technique.

The fenestration operation, on the other hand, is a detour procedure which is of great value in cases of irreversible ankylosis not amenable to stapes mobilization techniques. Since the surgical approach necessitates removal of the incus and the head of the malleus, the impedance matching action of the middle ear is usually lost, and the gain in hearing is not as great as that which can be expected in stapes mobilization. Nevertheless, in properly selected cases, it will improve the hearing threshold* to at least a 30-decibel level or better, which is adequate for the hearing of speech in most situations.

In general, any patient who has clinical otosclerosis with an adequate bone-air gap and a bone conduction level not lower than 45 decibels, may be considered a candidate for stapes mobilization. There are no age restrictions, and since the procedure is done under local anesthesia with very little surgical trauma, there are no major contra-indications medically.

There has been a significant evolution in the modus operandi of stapes mobilization since the first publication of Rosen[4] when he advised mobilization via the stapedial neck with a specially curved instrument. Shortly thereafter, other investigators[2] suggested application of force through the lenticular process of the incus, through the capitulum of the stapes, and other modifications in the region of the incudostapedial joint. It was quite logical that attempts to mobilize the ankylosed stapedial footplate should begin at the incudostapedial joint, because of its anatomic accessibility and the possibility of transmitting force through the stable crura. However, it was noted that incudostapedial dislocations (Figure 1) could be troublesome, and that crural (Figure 2) fractures could be severe problems. In a large number of cases, force applied to the joint itself could give a sensation of mobilization which in fact was mobility due to crural trauma. In such cases the stapedial footplate remained ankylosed even though the stapes seemed to be mobile on digital palpation. This approach, coupled with nonquantitative, nonaudiometric evaluation of adequacy of the surgical procedure, was at fault in many failures.

Through the recognition of the importance of quantitative measurements, and the utilization of a mathematical formulation of surgical physiology

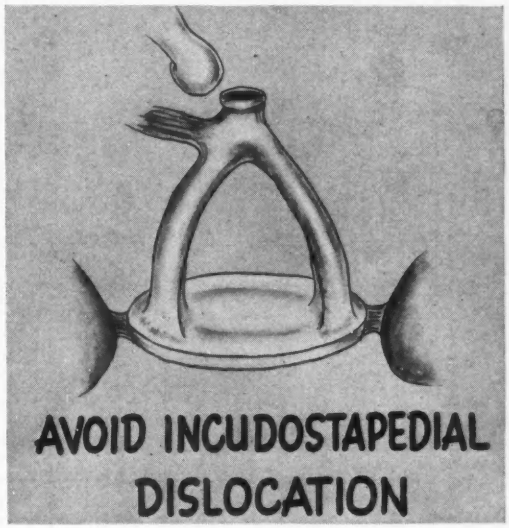

Figure 1

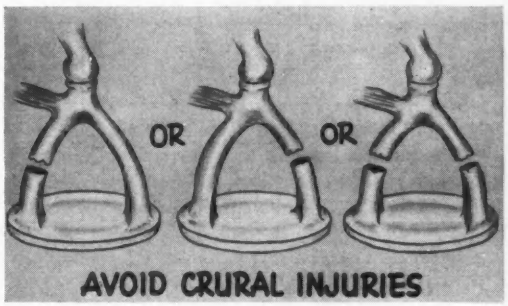

Figure 2

through the "audiometric nomograph"[3] technique, some of the dangers that were due to inadequacies in surgical manipulation were pointed out. Recognition of these inadequacies coupled with better magnification and illumination of the fossa ovalis and stapedial crura, logically turned the attention of otologic surgeons to the footplate region.

Methods of operative treatment of the footplate have been quite varied—of direct fractures, incisions, applications of hammer force, fenestration and peripheral curettage of the footplate region. Each of these procedures has its advocates who ascribe special merit to one or another technique. In all of these footplate approaches, there has been one area of agreement—namely, that preliminary operative results are greatly improved in numbers and in the degree of improvement in hearing.

I have previously stated[4,5,6] that the purpose of stapes mobilization should be lysis of the ankylosed stapedial footplate in such a manner that the entire footplate or a movable fragment will be in con-

*The hearing threshold is the least intensity of sound, in decibels, that the patient can hear.

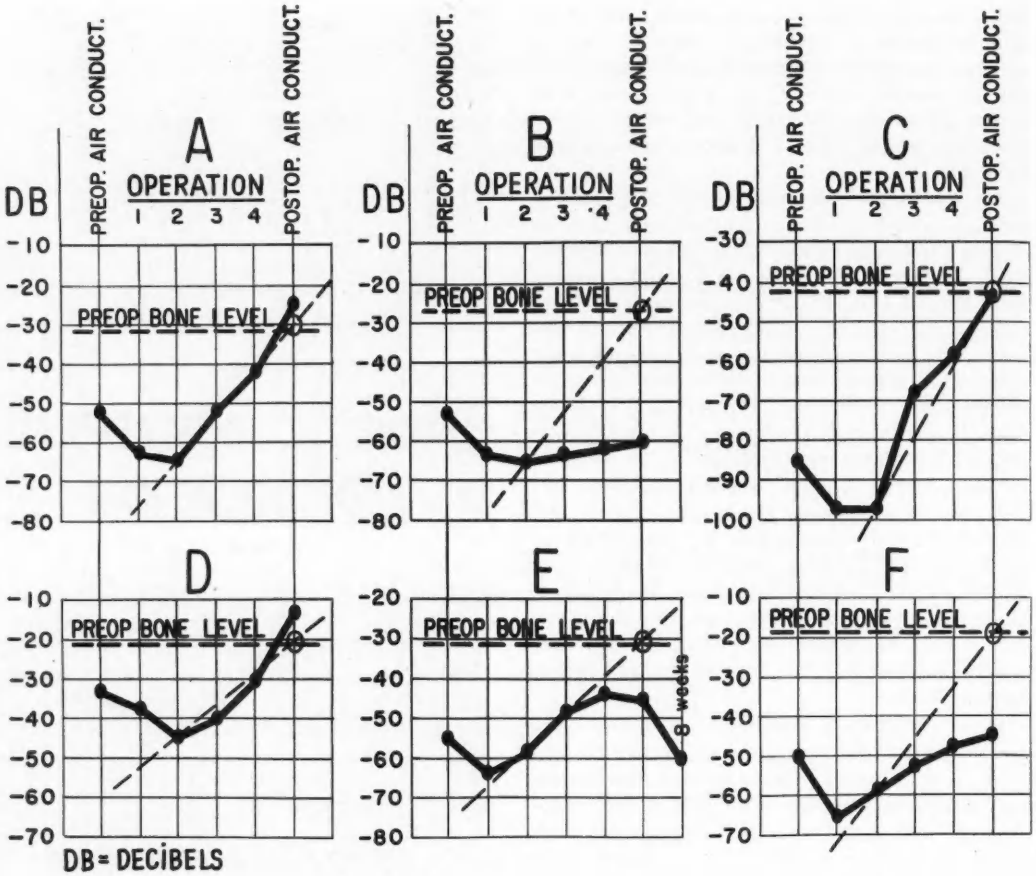

Chart 1.—Examples of results of audiometric testing from time to time during operation. The diagonal interrupted line represents the preoperative prediction. The horizontal numbers 1, 2, 3 and 4 represent various stages during the operation at which audiometric "sampling" was done to inform the surgeon how near or far he was from the desired effect. The charted results are examples of:

A, adequate stapes mobilization. Note that the dark line—showing the progress—conforms closely to the line of prediction.

B, failure to mobilize. Note that the progression line does not rise with the line of prediction.

C, adequate mobilization in a case of wide bone-air gap.

D, adequate mobilization in a case in which the bone-air gap was narrow.

E, adequate mobilization followed by early re-ankylosis of the footplate and loss of initial gain in hearing.

F, inadequate mobilization. Note that progression line shows a gain in hearing but does not conform to the predicted gain as represented by the interrupted diagonal line.

tact with the intact mobile ossicular chain, either through one or both stapedial crura, resulting therefore in either a monopod or bipod stapedolysis. This physiological objective remains a necessity.

In order to monitor the progress of applications of force to the stapediovestibular lesion, some method of physiological guidance appeared necessary. Since visual and digital information did not suffice for this purpose, the only method that could be relied upon was a "titration" based upon threshold changes as determined by audiometric studies carried out during operation.

Stapedolysis is not an all-or-none operation. True, an all-or-none approach will yield a certain number of successful results simply because of the nature of forces and resistances encountered, but a large number of failures resulted from the application of the all-or-none principle. The ossicular chain will not stand for all-or-none application of force, as is evidenced by the large number of crural fractures

Figure 3

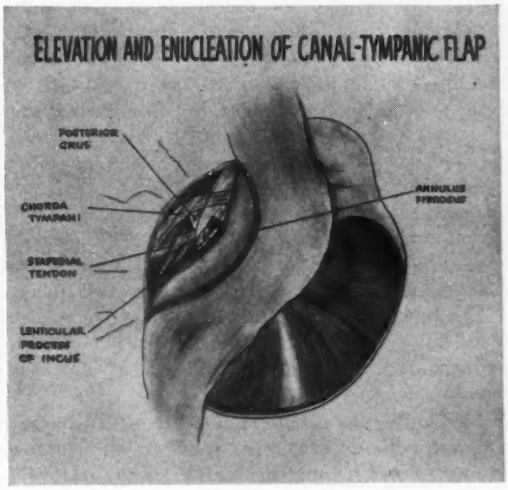

Figure 4

and incudostapedial disarticulations reported by several investigators.

Methodical degree by degree transmission of force sufficient to produce maximum lysis and yet insufficient to disrupt the middle ear mechanism is obviously a desideratum. This guided and titrated application of force cannot be based upon simple physical measurements of the amount of force applied. Serial determinations of the hearing threshold, using audiometry during operation, are required.

It was recognized some time ago that such serial measurements must be evaluated in each case on the basis of a ratio formulated from the bone-air gap. This led to the development of the nomograph technique, which has been described in several publications previously.[3,4] The nomograph, a graph constructed according to a mathematical rule, allows the plotting of progression of the surgical procedure on the operating table and offers the otologic surgeon precise guidance as to the adequacy of lysis. It also indicates ossicular discontinuity, inadvertent footplate fenestration, round window lesions, and inadequate lysis. By using an averaging technique developed by Fletcher,[1] the Fletcher formula gives us a single figure of merit for speech reception threshold which we call the *equivalent* SRT, and this single figure of merit allows for convenient mathematical comparisons. Chart 1 illustrates typical nomographic studies in a variety of conditions encountered surgically.

A monitored peribasal technique as used at present may be described as follows:

Premedication. Simple barbiturate premedication is adequate in most cases and the exact dosage is dependent upon the age, weight and general condition of the patient. Opiates are not generally used. It is desirable that the patient be calm and relaxed but not so sleepy that accurate audiometric studies will not be possible.

Skin Preparation. Routine surgical skin preparation is used after the hair has been carefully draped and covered by a stockinet cap. Aqueous Zephiran® (benzalkonium) is used in a 1:1000 solution within the canal for purposes of antisepsis and production of drum edema to allow easier enucleation and elevation of the canal tympanic flap.

Anesthesia. The cutaneous lining of the bony canal wall is adequately anesthetized by blocking with 2 per cent Xylocaine® (lidocaine) with epinephrine 1:20,000.

Incision and Elevation of the Canal Tympanic Flap. The procedure is performed through ear specula of various sizes. Illumination and magnification must be precise and are usually obtained through headlight-loupe combinations.

An angulated circumferential scalpel is used to create a fairly large posterior omega-shaped incision as illustrated in Figure 3. This skin periosteal flap is dissected down to the tympanic annulus and the drum insertion is elevated from the bony annulus so that the middle ear is exposed in its posterior one third (Figure 4). Sufficient bone is removed from the annular margin (Figure 5) to allow for adequate exposure of the lenticular process of the incus, the stapes, the chorda tympani, the stapedial tendon, the crura, the promontory and the round window niche. All of these structures must be visualized before surgical approaches can be made safely.

Palpation of Incus. The incus is gently tested for mobility (Figure 6) by palpation with a fine angulated needle probe engaged within the lenticular

periosteum. Even in the presence of a rigidly ankylosed stapes, there is a degree of mobility of the incus which is easily determined by such palpation. If the incudomalleolar joint is fixed, as it may be, in congenital anomalies or adhesive otitis media, this diagnosis is all-important. Such incudomalleolar fixation may well account, by itself, for the entire conductive loss; or, on the other hand, it may coexist with stapediovestibular fixation and cause failure if its presence is not recognized. (If incudomalleolar joint fixation is recognized, the stapes mobilization approach alone is usually inadequate.)

The integrity of the incudostapedial joint is then assessed by palpation. A loose joint will call for extraordinary care in manipulation in order to avoid incudostapedial dislocation.

Incudostapedial Manipulation. It has been my experience that rarely can transincudal or transcapitular force be used successfully in the relatively early or mild ankylotic states. It is not possible to tell, on the basis of the hearing threshold, the age of the patient or the duration of the disease whether there is only mild ankylosis. However, in many cases of the less mature forms of the disease, the ankylosis may be confined to the anterior limb of the footplate, or occasionally to the posterior limb alone. In such instances, safe lysis may be possible by force applied to the entire ossicular chain by way of the lenticular process or the capitulum. The former is preferable, since it has a slightly lesser tendency to dislocate the joint. However, if the joint is loose to begin with, it is preferable to use force on the capitulum.

The digital force used must be firm but gentle, and while force is being applied the entire ossicular chain must be observed as a *total mechanism* with special attention to the footplate region. In most successful cases there will be a general loosening and the stapes will usually be displaced slightly in a posterior direction. When effective displacement appears to have occurred, an audiometric test with nomograph guidance is obtained. Such alternations of application of force and audiometric tests are used until a decision is reached that the optimum result has been attained.

Peribasal Technique. In most instances, the approach I use is directly to the peribasal region, and not to the footplate per se. The first step is a careful survey of the footplate and contiguous areas. If such a study reveals, for example, an advanced invasion of the anterior crus and anterior limb of the footplate by the otosclerotic process, attempts at bipodal (two-legged) lysis are almost certainly doomed to failure. In such a case, the only hope of success is in monopodal (one-legged) lysis with anterior crurotomy. If, on the other hand, inspection reveals normal integrity of both crura but invasion of the

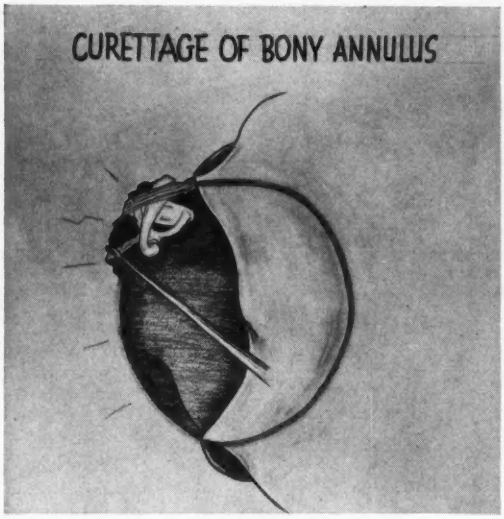

Figure 5

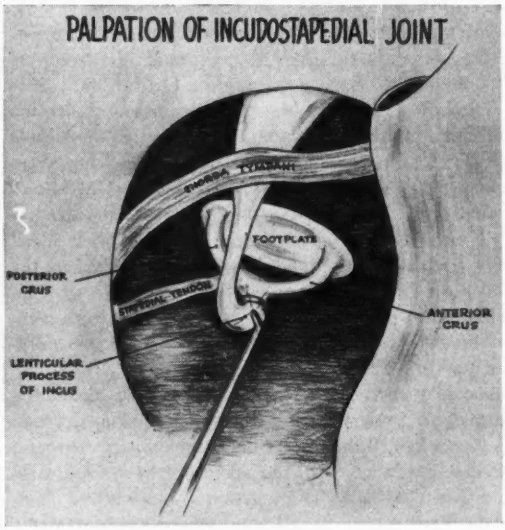

Figure 6

footplate and ligament by otosclerotic bone, *peribasal* use of a needle probe can be highly successful and result in preservation of the anatomic continuity of both crura (bipodal with a mobile footplate)—a normal, and thus desirable, physiologic state. To achieve careful footplate lysis with attention to the foregoing physiologic principles, force can be applied digitally with needle probes of varying sizes and angles, and tiny chisels. Such instruments may also be used in severe ankylosis problems, in conjunction with an electric hammer or microvibrator.

The survey of the footplate and contiguous area,

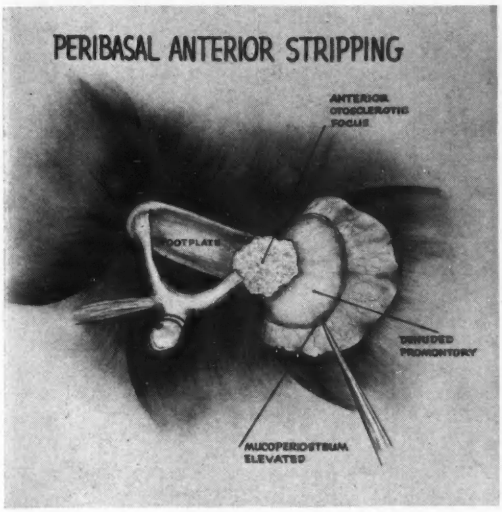

Figure 7

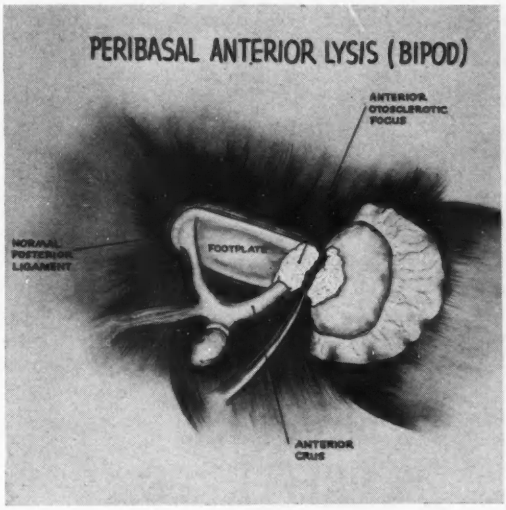

Figure 8

as referred to above, is frequently hampered by a pathologic thickening of the mucoperiosteum in the anterior crural area. Extensive otosclerotic invasion of the anterior crus, the anterior half of the footplate and the anterior aspect of the oval window niche will be accompanied by a thickening of mucoperiosteum in this area, making accurate viewing impossible. Consequently, it is necessary to strip the mucoperiosteum from this region as illustrated in Figure 7. With a small needle probe or a tiny curet, a small incision is made in the mucoperiosteum along the inferior margin of the footplate region and extending anterior to the anterior crus. This mucoperiosteal incision is carried down to the promontory bone, and the mucoperiosteum is reflected anteriorly and inferiorly away from the oval window niche. Frequently this will permit a surprisingly good view of the gross otosclerotic lesion and will enable more precise application of instruments to the peribasal region.

In using the peribasal technique to attempt bipodal lysis, it is desirable to insert the needle probe (Figure 7) first immediately anterior to the anterior crus and toward the vestibule through the region normally occupied by the annular ligament, which is frequently replaced by pathologic bony growth (Figure 8). If the disease is limited to the anterior limb, this maneuver alone may be adequate to produce lysis.

If adequate lysis is not demonstrated visually and confirmed nomographically the same procedure is carried out in a similar position posterior to the posterior crus, again through the region of the annular ligament, since in such cases there will be a lesion posteriorly as well as anteriorly (Figure 9).

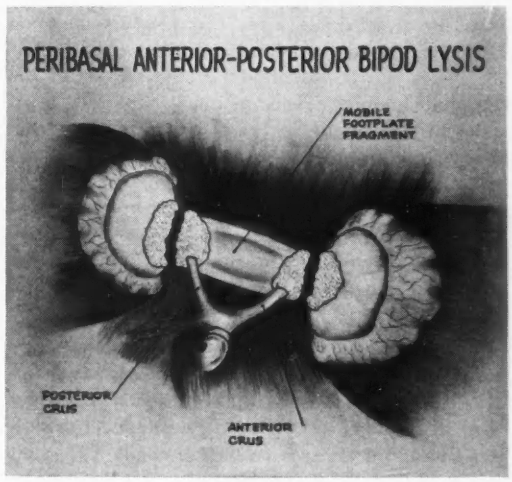

Figure 9

In general, bipodal lysis (Figure 10) as described above, seems more successful than monopodal lysis. This is to be expected because of the more advanced degree of disease in the cases in which monopodal lysis is done. Nevertheless, monopodal lysis has brought about satisfactory salvage of hearing in enough cases to justify use of the technique where necessary (Figure 11).

Direct fenestration of the footplate itself has been advocated by some investigators, but it appears to be an unphysiological procedure and I have not obtained satisfactory results with it.

When nomographic studies indicate adequate lysis of the ankylosis with preservation of ossicular and drum membrane continuity, applications of

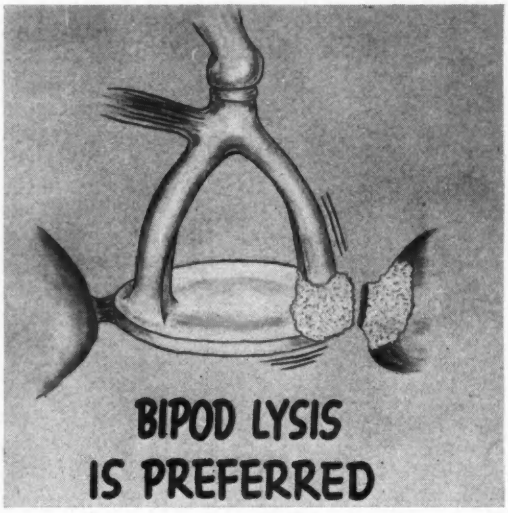

Figure 10

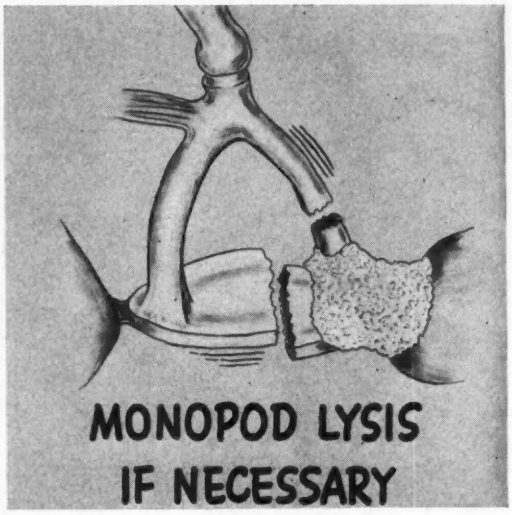

Figure 11

force are stopped and a few tiny pieces of gelfoam saturated with neohydeltrasol solution (Figure 12) are placed circumferentially in the region of the stapedial footplate. This gelfoam barricade is of value in sealing off any minute labyrinthine fractures and in protecting the perilymph space from the full impact of transtympanic airborne sound. Tests then made by surgical audiometry are more likely to be reliable and the possibility of spurious direct acoustic transmission to the perilymph can be avoided. The neohydeltrasol solution has caused no undue reactions.

Closure and Packing. The canal tympanic flap is gently replaced and carefully moved back into position. If there has been a perforation or marginal tear, it is closed by a small, full-thickness skin periosteal graft taken from the ear canal. The canal tympanic flap is covered with four or five rayon strips saturated in normal saline solution, and a few cellulose sponge pledgets saturated with cortisone ointment are placed within the canal to exert gentle pressure on the rayon gauze and on the canal tympanic flap. Such packing appears advantageous in preventing undue postoperative capillary oozing and in keeping adequate tension on the drum so that it heals with normal physiologic restoration. The ear canal is filled with cotton and a mastoid dressing applied for one day.

Postoperative Care. The patient remains in the hospital overnight, and is usually discharged the following morning unless there is unusual vertigo, which occasionally occurs. Antibiotic therapy is used on all patients, and a broad-spectrum drug is usually chosen after careful inquiry as to previous

Figure 12

history of drug sensitization. Such prophylactic therapy is continued for five to seven days.

Vertigo occurs in about 30 per cent of patients. Usually it is utricular in nature. In most cases it is abated by giving dimenhydrinate (Dramamine®), and usually it is self-limited. In general, vertigo is less of a problem with stapes mobilization than it is in fenestration operations.

The packing is usually removed on the sixth day, following which no special measures are necessary. By that time, the skin has usually healed and edema of the drum is subsiding. The hearing usually is somewhat impaired by the packing but becomes more acute upon removal of the packing and then improves further in the ensuing week or two. De-

layed shifts in the hearing threshold may occur in some cases due to hemorrhage or temporary interferences with the coupling mechanism of the ossicular chain.

Complications. No serious complications occurred in almost 700 cases. Tympanic perforation occurred in two instances. In a few cases loss of hearing increased after operation, owing to reankylosis, but there were no cases of cochlear destruction. Two patients had persistent mild utricular vertigo.

Reankylosis. Since the surgical operation is directed to the otosclerotic lesion per se, it would be expected that osseous regeneration would produce reankylosis. This does occur in some cases, sometimes soon after operation, sometimes after a considerable interval. The exact incidence of reankylosis cannot be stated at this early stage in the use of the stapes mobilization operation, but it does not appear to be higher than closure of the fenestra in fenestration. It probably develops in not more than 15 per cent of cases in which the operation is at first successful.

Reoperation is merited in cases of reankylosis. In the series here reported upon, successful results were obtained in the revisionary operations about as often as in the primary procedures. If, however, reankylosis occurs again, further revision would be justified in selected cases only. In most such cases, fenestration is indicated.

Tinnitus. In my experience, the tinnitus associated with otosclerosis disappears in most cases of successful mobilization, and persists in cases in which the operation fails. There are, however, exceptions in both categories.

Binaural Stapedolysis. Since speech audiometric studies have shown that discrimination (intelligibility) scores are considerably higher when hearing is binaural, and since binaural hearing also makes possible directional acoustic perception, it seems advisable to do binaural mobilization wherever possible. Not always, however, is the result in the contralateral ear the same as that obtained in the first ear operated upon.

Perspectives.

A. Recently this procedure has been extended to patients who do not have normal tympanic membranes by combining stapes mobilization with myringoplasty in a one-stage operation. The specific technique has been reported elsewhere.[6]

B. A conductive efficiency evaluation concept has recently been proposed by A. L. Holcomb, my research colleague, who suggests the utilization of the formula derived by dividing the bone-air gap into the decibel gain. The resulting percentage gives a reasonable measure of the conductive efficiency in restoring impedance-matching function to the middle ear or in removal of the conductive block.

Results. Postoperative results have been reported in great detail in previous papers.[4,5] The results reported are determined by appraisal of the first tests carried out approximately two weeks after operation. The terms here employed are the same as those used in previous reports, and may be defined as follows:

A. "*Thresholds.*" The values of acoustic threshold are stated in terms of the equivalent speech reception threshold derived from the Fletcher formula.* Thus, since audiometric measurements vary in 5-decibel steps, the equivalent S.R.T. values used here usually vary in 2.5-decibel steps.

B. "*No Change.*" The term *no change* is used to indicate a postoperative air conduction level that has not changed by more than 5 decibels, plus or minus, from the preoperative level.

C. "*Losses.*" If the air conduction level after operation is 7.5 decibels or more below the level before operation, the decrease is described as "loss."

D. "*Partial Gains.*" Partial gains are increases of 7.5 decibels or more in air conduction perception, but less than those defined as success.

E. "*Success.*" Dual criteria must still be retained for success, since most other investigators who report on the subject are still using the fenestration criteria under which any postoperative level within 30 decibels of normal is considered successful. However, I am convinced that removal of the air conduction block (or elimination of the bone-air gap) should also be considered a "success" regardless of the final air conduction level. Since audiometric measurements are not considered accurate in measurements finer than 5 decibels, I arbitrarily assume this criterion: If air conduction after operation is not more than 7.5 decibels below preoperative bone conduction, the operation is considered successful. (In general the only confusion likely to occur from this dual definition of success lies in the region in which air conduction is between 30 and 40 decibels and bone conduction is near normal. Most of the successful cases, as judged by the 30 decibels standard only, lie in this area; and counting them as successful materially improves the results reported for such groups. Approximately 43 per cent of the successes reported qualify in both categories; the remainder are about equally divided between cases that qualify in one category only.)

In the first 500 cases in the series here reported upon, successful result as judged by our dual standard was obtained in 52 per cent of cases. In 43 per cent of cases, results qualified as successes in both categories. In approximately 24 per cent of the 500

*An average of the two best threshold responses at 500, 1,000 and 2,000 cycles.

cases, results were eventually classed as failures for various reasons. In another 24 per cent, significant gains were obtained, varying from improvement of 7.5 decibels to as much as 40 decibels, although not qualifying as successes in either of the stated categories.

In the most recent group of 100 cases in a study of 600 cases, results were successful in 62 per cent; in 29 per cent significant but less than successful gains were obtained; and in 9 per cent the operation was a failure.

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Social Security Footnotes

THE GOVERNMENT, while slow to acknowledge anything wrong with the Social Security system, underestimated the demand for benefits. Women who could obtain benefits at 62, 63, and 64 decided to do so even if the payments were less than they would be at 65. Farmers suddenly turned out to be older than expected. Some began to pay social security taxes on reported income of $4,200 which exceeded their income in prior years. Then they applied for benefits after paying taxes for six quarters. Many people who had retired and were well beyond 65 years of age, dug up jobs for themselves and paid social security taxes for 18 months, thereby qualifying for benefits of from $30 to $108.50 monthly for life. Social security experts in making their cost projections underestimated the ingeniousness of the American people when federal give-aways are as widely advertised as are social security benefits.

—From the Department of Public Relations, American Medical Association

The Treatment of Diabetes Mellitus

WILLIAM W. H. POTE, JR., M.D., Los Angeles

• **The treatment of diabetes mellitus continues to challenge all physicians regardless of special interest. Although there have been many advances, treatment still primarily centers around education of the patient, particularly in regard to proper balance of diet, insulin and exercise. The exact role of the new oral insulin substitutes is not yet fully established. With proper consideration of the fundamentals of treatment and team-approach, there can be life added to the years and years to the lives of our increasing diabetic population.**

THE TREATMENT of diabetes mellitus presents a challenge to all physicians. Each physician must develop a philosophy regarding the treatment of diabetes if he is to carry out his responsibility to his patients.

The syndrome known as diabetes mellitus represents a very active and changing process, often acute. It crosses all areas of medicine. Many metabolic and endocrine processes are affected by diabetes. In no other condition is the arteriosclerotic process more evident, more accelerated than in diabetes. It matters not how specialized a physician may become, if diabetes develops in a patient under his care, he must consider special problems which may follow.

One of the most important contributions to the detection of diabetes has been the use of the routine urinalysis on every new patient, although glycosuria so detected is not diagnostic, nor is a negative result reliable in an older patient with an elevated renal threshold. Blood sugar determination is being used much more frequently today; and there is less insistence upon determination of fasting blood sugar, for it is recognized that blood sugar determined at any time, if interpreted in relation to the time of food intake and accompanied by glycosuria, may aid in the diagnosis of diabetes.

Even though today there is much more information available both for patients and physicians, too often diabetes is not properly dealt with. The patient has fear and is annoyed by the inconvenience of the necessarily imposed regularity of his life. Instead of developing and following a planned program, he too often resents, rejects, ignores or neglects the disease. Moreover, sometimes when the patient needs a friend to help him understand the treatment and the fact that he can live a normal life, his physician is not ready to explain diabetes to him in an understanding way. It takes time. There is much detail. There is much controversy. The patient is not given a clear-cut approach to diabetes. If the physician does not have the goal clearly in his mind, the patient is left confused and should not be expected to take care of himself. Obtaining acceptance of the treatment program is the physician's responsibility. If each physician is to meet the challenge, he must accept certain goals for the over-all problem of diabetes and certain specific goals for an individual patient.

The general goals in the treatment of diabetes are as follows: (1) To prevent death; (2) to prevent or delay sequelae; (3) to restore the metabolism to normal; (4) to help the patient to find a balanced program; (5) to detect early diabetes; (6) to prevent diabetes.

There should be specific goals in the treatment of individual patients. Diabetes should be controlled even if it cannot be cured. Physiological chemical values (blood sugar 100 mg. per 100 cc. fasting and 150 mg. per 100 cc. after meals) can be obtained in many patients without undue hardship to the patient and without incapacitating or significant insulin reactions. The patient can become aglycosuric or at most excrete in the urine not more than 5 to 10 per cent of his carbohydrate intake in 24 hours.

The patient must be assisted to function effectively at home, at work and in the community. It is desirable for him to maintain a stable weight, preferably normal or 5 to 10 per cent underweight. At present physiological control appears to be the best approach to prevention of sequelae.

To achieve these goals there are certain aids: Diet, insulin, exercise and education. More recently oral-hypoglycemic drugs have been under investigation to determine what contribution they can make to the care of patients with diabetes.

Diet

Diet therapy has changed greatly since the undernutrition era. As far as quality is concerned, a diabetic person can eat the same food as one who

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TABLE 1.—*Diet—Exchange Values*

Exchange List	Carbohydrates (gm.)	Protein (gm.)	Fat (gm.)	Calories	Amount
List 1: Milk	12	8	10	170	1 cup whole
List 2A: Vegetables	negligible				Limit 1 cup cooked
List 2B: Vegetables	7	2	0	35	½ cup
List 3: Fruit	10	0	0	40	.....
List 4: Bread	15	2	0	70	½ cup
List 5: Meat	0	7	5	75	1 oz.
List 6: Fat	0	0	5	45	1 tsp.

does not have the disease. It is most important that the patient eat specific amounts at specific times and at stated intervals, particularly if he is taking insulin. Patients' food patterns differ. The diet should be fitted to the family habits of eating. More than half of diabetic patients could obtain physiological control if they but followed a diet carefully. Weight reduction is impossible unless there is an adequate caloric deficit. Being the most easily quantitated portion of treatment, the diet is still the most basic part of the treatment of diabetes. Weighing of food is not necessary in all cases, although doing so is good as a training procedure for patients. The trained eye and conscience make it easier to follow a specific diet. For patients who are prone to hypoglycemia and ketosis, when one gives multiple doses of insulin it is probably important to weigh the high-carbohydrate foods and the meat.

For the majority of patients the "exchange diets" are probably satisfactory if understood and followed. It is certainly easy for the patients to visualize the food and to understand substitution. It is easy for physicians to remember the common values in the exchange system. If one remembers the values given to each of the six types of food and the amounts usually used, it is easy to calculate the values of food that one is eating. One can easily change the diet to fit his needs and desires (see Table 1).

Insulin (Table 2)

Insulin should always be used if the patient is severely symptomatic, or has infectious disease or if immediate control is desirable. If the patient is asymptomatic and nonketonuric, and is heavy, insulin should be withheld, since insulin therapy is not conducive to weight reduction. Insulin therapy has become more complex in recent years; there are at least five kinds of insulins for use in various situations (Table 2). Regular insulin has now been removed from the market and has been replaced by crystalline-zinc insulin (CZI) with a peak action of three to five hours in most patients. Crystalline-zinc insulin should be used primarily for an emergency such as coma or infection and as a supplementary insulin. One patient, recently observed, who had taken insulin three times a day since 1935, found that it was much easier to adjust insulin therapy, food and exercise when he took divided doses of CZI rather than long-acting insulin. This patient has had diabetes 21 years yet had only some microaneurysms in the eyes. A twin brother, who never followed a careful program, died recently with intercapillary glomerulosclerosis.

Protamine-zinc insulin was the insulin in general use for many years, but it has a prolonged effect making it inadequate for daytime control, as the greatest effect is when it is least needed.

Globin, NPH and lente insulin are effective intermediate insulins. NPH and lente insulin appear to have slightly more prolonged effect than globin. With use of these insulins, meal distribution should be in thirds with a mid-afternoon feeding in addition. Crystalline-zinc insulin may be added readily to NPH to control the after-breakfast spill, which in many patients is not accomplished with a single dose of NPH. In still other patients it is necessary to give NPH at bedtime in small amounts to achieve night-time control. For immediate effect, crystalline-zinc insulin may also be added to lente. Most patients will be well controlled with a small amount of crystalline-zinc added each morning to whatever intermediate insulin they are using. The guide for the dosage of intermediate insulin, of course, would be the before-breakfast urine sugar, and for the crystalline-zinc would be the before-lunch urine sugar. Only a fresh specimen of urine, voided after the bladder is emptied of urine that has accumulated over a long period, should be used for determination of sugar content as a guide to change in insulin dosage. The urine sugar is a good guide to diabetic control if it is regularly tested and the patient's renal threshold is known. It is now possible to obtain ultra or long-acting lente and semi or short-acting lente. With these it may be possible to use only lente insulins.

In taking insulin of any kind, one should keep certain points in mind: Probably insulin atrophy would be seen less frequently if the patient varied the site of injection, which can be done easily if a record of injections is kept on a map. Many patients

TABLE 2.—*Properties and Uses of Various Kinds of Insulin*

Type of Insulin	Action			Indications	Precautions
	Onset	Peak	Duration		
Rapidly acting:					
Crystalline-zinc..	½ to 1 hr.	3 to 5 hr.	6 to 8 (12)* hr.	Emergencies as coma, infection, operations; supplementary	Eat soon after injection; equal meals equally spaced.
Intermediate acting:					
Globin.....	1 to 2 hr.	6 to 11 hr.	15 (24) hr.	Daytime glycosuria	Take mid-afternoon food.
NPH.....	2 hr.	7 to 10 (10 to 20) hr.	28 to 30 hr.	Daytime glycosuria; basic insulin	Meal distribution: One-thirds, with mid-afternoon feeding. Increase dose slowly.
Lente.....	2 hr.	7 to 10 (10 to 20) hr.	28 to 30 hr.	Daytime glycosuria; basic insulin	Not over 5 units every third day. CZI may be added.
Prolonged-acting and protamine-zinc					

*Parentheses indicates duration cited by some investigators.

complain of allergic reaction to insulin, particularly when they first take the drug. However, the red elevation at the injection site may be due to too superficial injection or to irritation from alcohol. It is important to inject insulin deep enough. If the patient at first has allergic sensitivity to insulin, he will probably become desensitized if insulin is continued. It is the patient who has taken insulin, discontinues it, and then begins again months or years later who develops severe sensitivity. If the patient is known to be allergic to insulin, there is an obligation to desensitize him because he may sooner or later have a complication or an infection leading to acidosis, which would necessitate use of the hormone. Many patients with hypoglycemia have strong rebound hyperglycemia, and too often in such circumstances additional insulin is given rather than having the amount reduced. Often the rebound glycosuria or the sugar content of residual urine masks the negative urine tests which should accompany the low blood sugar. If the program is well planned, very few patients will have frequent or severe insulin reactions. When they occur they should be treated promptly and with enough food.

Exercise

A diabetic patient should take the amount of exercise usual for persons of his age. It is best if his heaviest exercise can be taken at a planned time each day. If unusual, unplanned exercise is taken, about ten grams of carbohydrate per half-hour or hour of exercise should be ingested, the amount depending on how vigorous the exercise is. It is well recognized that exercise diminishes the requirement of insulin or increases its effectiveness. The usefulness of exercise in diabetic patients has not been fully explored. It should be borne in mind that whenever the program of the patient is altered, diet, insulin and exercise must all be adjusted.

Education

An understanding patient is the most cooperative patient. If the physician does not have the time nor interest to instruct the patient properly or does not have a trained aide who can do so, he should send the patient to someone who can give him rudimentary instruction in diabetes. Each patient should have instructions regarding diet, insulin, foot care, insulin reactions and the treatment of reactions, and the general problems of diabetic persons. With some personal instruction, the many good manuals available can help the patient considerably.

Other aids in the care of a diabetic patient include blood sugar tests, urinalysis and acetone tests. All patients should be carefully trained in techniques of testing urine for sugar and acetone. Testing with Benedict's solution is the cheapest method and still very good. Clinitest® is most frequently used. The newer Clinistixs,® Testape® and Testixs® have special usefulness when the patient is traveling or is at work or at school. One must be sure that the patient understands the colors involved in use of Testape and that he reads the results after a one-minute interval. It is important that a patient get used to one method of testing and use it regularly. All patients should test at least once daily, particularly after the heavy meal. A patient with unstable diabetes or one who is taking insulin should make a urine test two to four times daily. The quantitative determination of the sugar content of the urine is an excellent guide to significant glycosuria. An older patient often has a relatively high renal threshold

and does not realize that "negative" urine may have no significance whereas a "positive" test result would mean decided hyperglycemia. The renal threshold may change after control is obtained.

Drugs for Oral Use

As to the new oral hypoglycemic drugs, there is no conclusive answer as yet. Tolbutamide has no serious toxicity (in contrast to carbutamide, which was removed from clinical investigation). Regarding the sulfa drugs, it is generally agreed that they lower the blood sugar. It appears that the pancreas is necessary for their action. The consensus is that when sulfa drugs are used the effectiveness of insulin is increased, probably through stimulation of the beta cells of the pancreas. There has even been some suggestion that these drugs may enhance the action of endogenous insulin. Some investigators have noted evidence of decreased hepatic glucose output with the use of sulfa drugs, although this does not appear to be a major effect when used in ordinary dosage. There are many conflicting results in the experimental work to date, probably due primarily to varied conditions of the research, species difference and interpretation. If it be true that these drugs work through the insulin mechanism, it may take at least one to three years to know whether it is useful to stimulate the pancreas of diabetic persons. The possibility of exhaustion of the beta cell mechanism should be considered.

Clinical experience has shown that the drugs for oral use are much more effective in patients who were mature at the time diabetes began—that is, a patient over the age of 45, usually obese, taking less than 40 units of insulin daily and having had diabetes for five years or less. Selection of patients by specific tests has been attempted. One way is by using a loading test* of three grams of tolbutamide and observing the decrease in blood sugar after four hours, a good response being a decrease of 20 per cent or more. If the patient has been using a small amount of insulin, it should be omitted the day of the test. If he requires large amounts of insulin, regular insulin may be substituted for a day or two before the test. Not all patients who respond to the loading test get a good result with treatment, nor do all nonreactors fail to respond to treatment. Another suggested test is the response of the patient in regard to ketonuria after the cessation of insulin. If ketonuria occurs, tolbutamide is not likely to work.

In evaluating the clinical results in the use of the oral hypoglycemic agents, one must carefully evaluate the basic control prior to the use of the drug. It is possible that some patients in whom diabetes is fairly well controlled by diet alone may receive sufficient additional help to maintain better control. Certainly many patients who have favorable results with the drug would do well on diet alone if the diet were carefully followed.

In evaluating drugs such as these, short term and long term toxicity must be studied. It appears that tolbutamide is far less toxic than carbutamide and has no significant side effects. The average dose for maintenance has been 1 to 2 grams daily. A newer nonsulfonamide hypoglycemic agent is now under study.

In considering the treatment of diabetes, one should remember that diabetic patients generally can be separated into two groups—those who were mature at the time of onset and those in whom the disease began in the growing years. The former are stable and often respond to diet alone. It is the juvenile type of diabetes that is more often unstable or labile and requires the use of insulin, and often is very difficult to keep in balance. It is the relatively small group of patients with unstable, insulin-sensitive and ketosis-prone diabetes who present the greatest problem in the treatment of diabetes. With properly used insulin mixtures and multiple doses per day as well as a carefully quantitated and regulated diet, the disease can be stabilized in most of these patients. It may be necessary to give as much as six feedings a day and a diet higher in protein and fat for better stabilization. Certainly the difficulties in control should not cause a physician to allow a patient to completely abandon desirable goals in treatment.

Diabetes is no longer a tragedy, but mostly an inconvenience. Patients must be convinced of this and be given the support that they need in working out livable programs. Just as the patient rejects, resents, neglects and ignores diabetes, too often the physician may resent, reject, ignore or neglect a patient with diabetes. Often the patients are elderly and unemployable, of a low income group, often on pension, socially neglected and difficult to rehabilitate. They need the help of responsible physicians. The ideal control of diabetes in a patient can best be accomplished by a team consisting of a cooperative patient, an interested physician, a well-trained dietitian, a nurse, a social service worker, a laboratory technician, a pathologist and a chiropodist. Each has a vital part to play in accomplishing the total program of care of the diabetic. This total program includes not only adequate treatment but more active research in the causes of the problems encountered in diabetes.

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*_Loading Test._ Subject should be free from keto-acidosis. All insulin is discontinued for three days except for patients with large insulin requirements or frequent ketonuria in whom CZI is used intermittently as needed for control of any heavy glycosuria. On the test day a fasting blood sugar is drawn and 3 grams of tolbutamide given orally. The subject remains fasting for four hours at which time the blood sugar level is determined again. A hypoglycemic response of 20 per cent or more is favorable.

Recent Developments in Surgical Skin Planing

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• In surgical skin planing steel wire brushes have been largely replaced by the less hazardous diamond chip burs or "fraises" and serrated steel wheels.

In addition to acne pits and wrinkling, multiple actinic (senile) keratoses are an important indication for planing. Planing provides a nonscarring method for the treatment of existing keratoses, as well as a prophylaxis against skin cancer by replacing the sun-damaged, precancerous epidermis with new epidermal cells derived from the cutaneous adnexa (pilosebaceous and sweat gland units).

There are clinical landmarks indicating the depth of planing which can serve as a guide to the operator and can be correlated with microscopic findings.

The results of experiments on the comparative effects of refrigerants on animal and human skin indicate that human facial skin can tolerate considerable freezing with ethyl chloride or dichlorotetrafluoroethane (Freon 114) but that mixtures containing large proportions of the much colder dichlorodifluoromethane (Freon 12) may be undesirable. Refreezing an area of the skin in order to perform a more adequate planing is not considered hazardous.

The regeneration of the skin following planing has three components: Epidermal, adnexal and dermal. The cells of the epidermis and the adnexa are equipotential. A knowledge of the anatomy of the acne pit enables the operator to decide which pits can be benefited by planing and which should be excised before planing. The successful treatment of acne pits of the face by planing in patients having keloids elsewhere on the body is reported.

FURTHER EXPERIENCE in the technique of surgical planing has led to the virtual abandonment of the steel-bristled brush because of its great coefficient of friction when compared with its cutting power and dangerous tendency to seize tissue suddenly and "gouge" deeply and uncontrollably without warning, especially at the periphery of a given area where the tissue is not frozen. The lips and eyes are zones of particular concern to the operator when using the wire brush. Gauze or hairs also become entangled frequently, resulting similarly in loss of control and occasionally breaking the flexible cable-shaft of the machine.

Two types of abrading disc have proved to be preferable. The safest is composed of a metallic disc, the periphery of which is covered with securely embedded, sharp-pointed diamond or ruby chips.[4] These are termed *fraises* and they are available in several sizes and shapes and in varying degrees of coarseness of the chips. They are intended for cutting materials of great hardness and can withstand considerable such usage before becoming dulled. Such rugged construction is not necessary for surgical planing, but the inconvenience and cost of replacement are much less than with steel-bristled brushes, which usually become unusable after a few operations. A small pear-shaped diamond fraise is useful around the nasolabial folds and glabella. The jeweled fraises are capable of cutting to any reasonable depth, but only in an adequately frozen area. The fine chip diamond fraises available at first were not entirely adequate for deep planing, but the recently available coarser grade is more efficient, yet retains the safety feature of almost complete freedom from gouging or catching.

A stainless steel disc with a serrated cutting edge[4] has also been found useful for deeper cutting. These

Figure 1.—Jet-spray handpiece. The operator presses with thumb to release refrigerant spray carried from tank by plastic tubing. Area to be planed has been demarcated with gentian violet. Cotton glove is for traction. Spatter guard protects operator from abraded skin particles.

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Figure 2.—*Left:* Multiple senile keratoses (farmers' or sailors' skin) before planing. *Right,* six weeks after one planing.

are available in several diameters, widths and shapes. They are not quite as free of the tendency to seize tissue and gauze as the jeweled fraises and should therefore be used with more nearly the same caution as that necessary with a bristled brush.

The jet spray handpiece previously described[10] has been further improved and is obtainable (Figure 1.). Drawing its refrigerant liquid continuously from a tank containing more than needed even for a prolonged operation, it eliminates the time necessary for the operator to change from a hand-held can to the abrading tool and thus increases the convenience and efficiency of the freezing-planing procedure.

Devices to protect the operator from the spray of abraded skin particles and blood are now in general use. The jet spray handpiece has a spatter guard, and similar devices are available to clip to the conventional handpiece. Beirne and Beirne devised a guard which also serves to collect the particles of skin.[1] We have attempted to use the material collected on the spatter shield (obtained by planing nonfrozen skin) as grafts for clean granulating leg ulcers, but the results have been disappointing.

Although pitted acne scarring continues to be the major indication for planing, the value of the method in the treatment of single or multiple senile keratoses is also very great. It is a rapid method of removing the lesions adequately without producing the scarring that may result from some of the other destructive methods commonly used. It has a prophylactic effect as well, in that it replaces the epidermal cells that have been exposed to years of actinic damage with new cells derived from the adnexal structures. Thus, patients who have a hereditary predisposition toward multiple precancerous senile keratoses and skin cancer have been given, by prophylactic planing, a fresh start with a new epidermis. In time, new keratoses may develop, but a substantial delaying action has been achieved. Furthermore, they have been spared the "patchwork quilt" effect that frequently results from the destruction of crops of keratoses by conventional methods over a period of years. The improved, more youthful appearance of the new skin is an extra dividend gratefully accepted (Figure 2.). In this condition, the planing should be relatively superficial, since the objective is removal of the sun-damaged epidermis. Epstein[5] recently stressed the value of planing in the treatment of actinic and radiation keratoses,[5] although his recommendation that it be used in chronic radiodermatitis should perhaps be qualified somewhat. We would not hesitate to plane individual radiation keratoses, but we would urge great caution in planing extensive areas of chronic radiodermatitis unless there is evidence that the cutaneous adnexa are still present. We consider planing of extensive areas that are devoid of adnexa to be contraindicated, since the normal sources for epithelial regeneration are absent.

Fine and moderate wrinkling responds well to planing (Figure 3). The more recent use of the diamond fraise with its greater safety enables one to plane quite close to the eyes without undue danger, although it is an exacting procedure.

Another situation in which planing is a useful adjunct is in bevelling the sharp edges which may result from the destruction, for example, of a basal cell carcinoma by electrodesiccation and curettage. Furthermore, one need not wait until a depressed scar has formed in order to improve it by planing but, as suggested by Zimmerman,[14] freeze and plane the sharp borders immediately following the destruction of the lesion. Depressed linear traumatic scars respond well to planing (Figure 4).

Hypertrophic scars tend to recur following plan-

Figure 3.—*Left:* Moderate wrinkling (and acne pits) before planing. *Right:* Two months after one planing.

ing. Therefore, if feasible, they should be excised and sutured. If they are treated by planing, the use of x-ray may help prevent a hypertrophic recurrence.

By observation one can delineate five depths of planing which appear successively during the procedure. (Burks[2] wrote on depth of planing, but our observations are somewhat different from his.)

1. First the pigment vanishes, indicating that one has removed the major portion of the basal cell layer.

2. The second level is determined by observing myriads of yellowish macules, each less than one-half millimeter in diameter. This indicates that planing has progressed down into the upper dermis, making the pilosebaceous apparatus visible. Because of the shredding of dermal tissue and linear striations, this level is usually not identifiable when a wire brush is used, but it is readily seen on the much smoother abraded surface that is produced when the diamond fraise is used on a thoroughly frozen field.

3. As planing goes deeper, the surface appears studded with yellowish lobulated papules. These are the sebaceous glands which have been cut across and is the depth usually achieved in a moderately deep planing.

4. If the planing is carried deeper, decided resistance of the tissues will be encountered. This is primarily because the tissue at that depth is below the penetration of freezing (about 2 mm.), as was pointed out by Strauss and Kligman.[11] Of course, if refreezing is done at this level, further depth can be achieved with relative ease, and caution is in order.

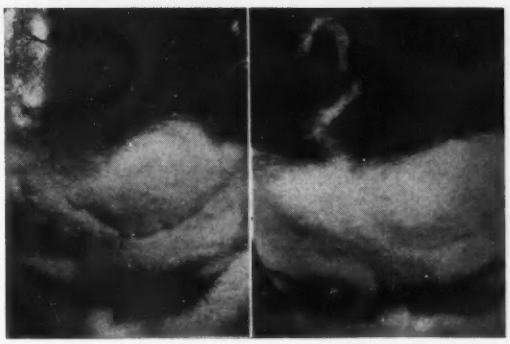

Figure 4.—Depressed linear traumatic scar. *Left,* before planing. *Right:* Six months after one planing.

5. Finally, if planing is carried to a still greater depth, yellow "nubbins" that are lobules of subcutaneous fat are exposed. This is well below the level of the sebaceous glands and at the depth of the deepest hair papillae. Planing to this level is very

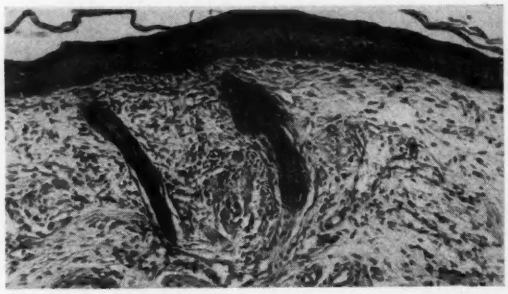

Figure 5.—Biopsy of facial skin two weeks after planing. Vellus (lanugo) hair follicles with primitive sebaceous gland buds are developing from the new epidermis. Rete ridges are still absent. Subepidermal portion of dermis appears composed of new collagen with young fibroblasts.

likely to result in removing so much adnexa as to cause scar formation.

Strauss and Kligman observed that the skin of the human cheek could be frozen for as long as 8 minutes with ethyl chloride or dichlorotetrafluoroethane (Freon 114, Frigiderm[12]) without producing severe damage, and that such reaction as did occur was no worse after 8 minutes than after one minute of application of the refrigerant.[11] They postulated that the great vascularity of the superficial portion of the skin prevents deep freezing and that the frozen surface itself acts as an insulator, preventing deeper penetration of the cold. In support of this, they noted that even heavily frozen skin was not frozen deeper than about 2 mm., as evidenced by the presence of freely bleeding, nonfrozen skin below this depth when planed quickly and forcefully.

In experiments we carried out with freezing the skin of the backs of dogs, significant damage was observed beginning after about one minute of application of either ethyl chloride or Freon 114 (without planing), and somewhat greater damage after one and a half and two minutes.[9] Differences between human facial skin and that of the dog's back probably account for these quantitative differences. Dichlorodifluoromethane (Freon 12) produced damage after 15 seconds comparable to that produced by ethyl chloride or Freon 114 after one and a half or two minutes. The difference is apparently directly related to the much colder skin temperature produced by Freon 12: (Ethyl chloride, —30° C.; Freon 114, —36° C.; Freon 12, —60° C.).

Experiments with mixtures of varying proportions of Freon 114 and the much colder Freon 12 have shown evidence of improved freezing capacity as compared with Freon 114 alone. However, we believe the proportion of Freon 12 must be kept relatively small (about 30 per cent) to avoid overfreezing, and the danger of refrigeration necrosis.[13] For

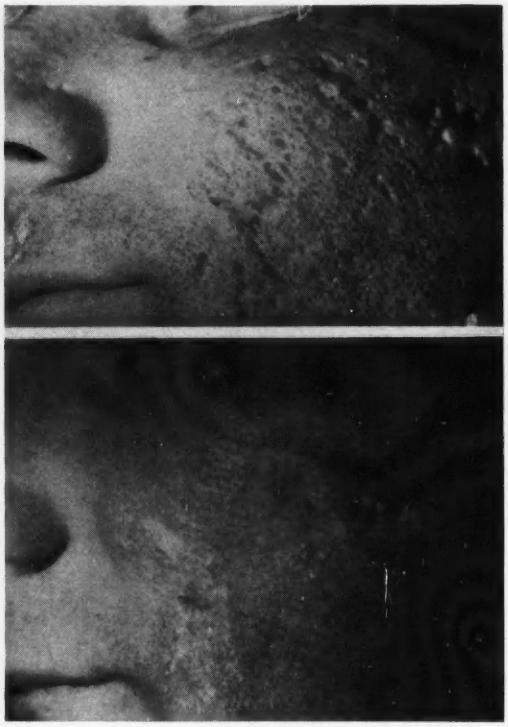

Figure 6.—Deep acne pits. *Upper:* Before combined excision and planing. *Lower:* After excision of several of the deepest pits, followed by planing of the balance.

example, a mixture of 43 parts Freon 114 and 57 parts Freon 12 was compared with pure Freon 114 by spraying on the back of the hand for 20 seconds. The colder mixture (skin temperature —55° C.) produced a considerably greater reaction than Freon 114 alone (skin temperature —36° C.).

We are in complete agreement with Strauss' and Kligman's observation that refreezing an area is not harmful.[11] It is our belief, supported by clinical observation, that refreezing an area when necessary in order to plane it more adequately at a given session is not hazardous and is preferable to the alternative of increasing the number of planing sessions. It is often preferable to continue planing another area while waiting until bleeding has stopped in the area considered to have been inadequately planed. After a few minutes it is much easier to refreeze this area without interference from the oozing of blood.

The regeneration of the epidermis from the remaining portions of the cutaneous adnexa within a week following planing is now a familiar concept.[3,9]

The milia which commonly occur three to six weeks after planing are actually "tiny cysts lined by stratified epithelium . . . and containing concentric lamellae of keratin."[6] They arise from epidermal

and adnexal cells which have been isolated in the dermis at the time of planing. These cells first dedifferentiate, and if they fail to rejoin the adnexa or surface epidermis, they then differentiate into milia. Moreover, the epidermal and adnexal cells are all equipotential, as evidenced by the rapid regeneration of new epidermis from every epithelial structure that remains after planing. All these specialized cells produce tongues of identical undifferentiated proliferating cells, which form a new epidermis; and from this, in turn, new vellus (lanugo) hair follicles and sebaceous glands regenerate.[6] The sebaceous glands are first evident as primitive structures between two and four weeks after planing and are mature and functioning by two months[6] (Figure 5).

We have been interested not only in epidermal and adnexal but also in dermal regeneration. Two weeks after planing there is a sharply defined subepidermal zone which is paler staining than the rest of the dermis below it. It has a loose, reticular appearance and contains many young fibroblasts, thus clearly suggesting that new collagen has been laid down during and after re-epithelization (Figure 5).

It is our experience that following a planing of moderate or greater depth there is not a restoration of dermal tissue equal in thickness to that removed; hence, the skin is actually a little thinner than before planing. However, the fact that there is at least partial restoration of the dermis seems to explain the clinical observation that even though there may be only a slight dell remaining as the bottom of a pit immediately after planing, there may be a pit of considerable depth reappearing after regeneration has occurred in a week or two. Since, in this situation, the floor of the pit with its epidermal lining has not been removed, dermal regeneration takes place on all sides of it, and the result of this particular planing session is only a partial decrease in depth of the pit. On the other hand, if the floor of this pit with its epidermal covering is completely removed by planing, then the resulting dermal regeneration will be uniform over the area, and there will be little or no evidence of the pit. The routine use of gentian violet[7] is of great value in visualizing the depth of pits, but some pits are too deep for complete removal by planing.

It is our practice to remove, either by elliptical excision and suturing or by a punch, those pits which appear obviously too deep for safe removal by planing. This is done three or more weeks before planing, and any suture marks resulting can then be planed along with the pits (Figure 6).

Our measurements of the thickness of biopsies of facial skin showed variations between 2 mm. and 4.5 mm., the skin being thickest on the cheeks and thinnest on the anterolateral aspects of the neck and adjacent to the eyes. When Strauss and Kligman performed what they considered to be very deep planing, the deepest removal of tissue was 2.0 to 2.5 mm., which is about half the total thickness of thick skin.[11] Their measurements and ours showed the average acne pit to be 1 mm. or less in depth, those considered as quite deep were about 2 mm., and only the very deep "ice pick" pits were deeper than 2 mm.

Figure 7.—Biopsies of "ice-pick" acne pits excised from bearded area of cheek of patient shown in Figure 6. *Upper:* Deep, narrow pit (2.7 mm.). *Lower:* Pit 1.8 mm. deep, with lateral extension and epidermal proliferation.

Microscopic examination of "ice pick" pits showed the extreme depth to which these scars can extend and the undermining or tunneling that can occur (Figure 7), although this may not be evident clinically.

Whether acne pits of average size and depth are removed completely or only partially at a given planing depends on the depth of the pit and the depth of the planing. Without excessively deep planing it may be impossible to entirely remove very deep pits with steep, narrow sides. This is a relative matter, depending not only on the depth of the pit but on its depth in relation to the total thickness of the dermis and its contained adnexa. For example, it may be possible to eliminate an "ice pick" scar on the thick skin of the medial or midcheek with safety, whereas the same depth of planing over the angle or ramus of the mandible or side of the neck could cause scarring due to complete destruction of the adnexa. Great care should be exercised to avoid excessively deep planing in the thin-skinned areas. This can best be done by not freezing to a board-like hardness in these areas and by planing lightly.

It has been our contention that planing of the face is not absolutely contraindicated in the case of a person of keloidal diathesis. This is borne out to a limited extent by the experience of one of us (Ayres), who has performed five full-face planings on three patients with pitted acne scars who had typical keloids elsewhere on the body (presternal area and arms). In each case a small test area was first planed on the face; then, no untoward reaction having occurred in six weeks, the full face was planed. One of the patients had very deep pits necessitating two deep planings. All three patients had an entirely satisfactory course over a period of many months of observation after planing.

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No. 1 Killer

The influence of fear as a deterrent to reckless driving seems to have been overrated, to judge by the comeback of cigarette sales after the lung cancer scare. But in Connecticut, where drivers lose their operator's license for 30 days for the first speeding conviction, fatalities were reduced 14 per cent. Governor Ribicoff said, "If people won't slow down to save their *lives*, they will to save their *licenses*."

Alcoholism

The Chronological Background Leading to California's Present Program

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• **An ancient problem is receiving modern attention in California. In this paper, the author briefly reviews the historical background that has made possible newer attitudes and study approaches to the problem of alcoholism.**

The chain of circumstances (and its welders) leading to California's broadly programmed attack upon alcoholism and its related complications is described.

THE HISTORICAL PROMINENCE of alcoholism has been recorded in the literature of every known civilization from antiquity. Philosophers, scientists, religious leaders and writers have observed and discussed man's relation to alcohol—that inevitable by-product of man's mastery of agriculture[19]—in terms variously laudatory and derogatory.

Biblical quotations (71 in all) relating to the use of alcoholic beverages are favored sources for argumentation by both "wets" and "drys," as are quotations from Shakespeare.

The Babylonian Code of Hammurabi, the oldest known system of codified law (c. 2225 B.C.), devotes several sections to regulation of the sale of alcoholic beverages in an attempt to control intemperate use of such beverages.[32]

The Chinese, whose traditional lack of alcoholic problems has provoked much study[3,20,21] even into modern times,[4] established and repealed laws against the manufacture, sale and drinking of wine no less than 41 times in the 2,500 years beginning about 1100 B.C.[29]

Many accurate and currently applicable descriptions of both the acute and chronic stages of alcoholism can be found in the writings of the Graeco-Roman era. Pliny the Elder (23-79 A.D.) wrote of the acute stage, and Seneca (5 B.C.-65 A.D.) in describing chronic alcoholism wrote: "The results are pallor, quivering of the muscles soaked in wine, and an emaciation due to indigestion and not to hunger. Hence the uncertain and tottering gait, and constant stumbling as if they were actually drunk; hence the swelling of the skin and distension of the belly, which has taken more than it can hold; hence the jaundiced and discoloured complexion . . . and the nerves dulled and without feeling or, on the other hand, constantly twitching. Why need I speak of the giddiness or the disturbance of vision and hearing, and the insidious pains in the head?"[33] The Dark and Medieval Ages, with their many conflicting beliefs and practices, donated to the future a rich, if confusing, mythology about alcohol, strong remnants of which persist even today.

National and international temperance movements and organizations for almost two centuries have been recognized forces in concerted attempts to deal with alcoholism. Similarly, insightful scientists, clinicians, lay bodies and individuals have constantly applied thought and effort to the solution of the problem.

A British physician, Doctor Thomas Trotter, in a thesis submitted to the University of Edinburgh in 1778, described drunkenness as a "disease produced by a remote cause"; and as early as 1830 in this country the Connecticut Medical Society emphasized the need for special hospitals for the treatment of this "disease."[1]

In 1885 the first meeting of the International Congress on Alcoholism was held in Antwerp.[25]

The League of Nations in 1926 suggested that alcoholism study be part of the League program, and encouraged enforcement of Article 22 of the League Covenant, which dealt with control of importation of alcohol into mandated colonies.[10]

Again in 1928, at the 13th Session of the League of Nations Health Committee, an active program on alcoholism was considered, but an evident lack of scientific and medical knowledge worked against the establishment of such a program.[47]

The United Nations, successor to the League of Nations, established its international health arm, the World Health Organization (WHO) as a functioning body in 1948. When the Expert Committee on Mental Health undertook the question of alcoholism in 1949, it recommended that the problems be studied by a special subcommittee.[55]

This Expert Committee on Alcohol and Alcoholism convened in first session in December, 1950,

Submitted September 3, 1957.

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and submitted as its basic recommendation: ". . . that WHO should take all steps within its power to stimulate public-health services, to undertake work on this problem and should be prepared to provide advisory, educational and other services on this subject to such national health authorities as request them."[56]

The alcoholism subcommittee classified the drug *alcohol* as "intermediate between addiction-producing and habit-forming drugs,"[59] and defined alcoholics as: ". . . those excessive drinkers whose dependence upon alcohol has attained such a degree that it shows a noticeable mental disturbance or an interference with their bodily and mental health, their interpersonal relations, and their smooth social and economic functioning; or who show the prodromal signs of such developments. They therefore require treatment."[58]

This committee also proposed the following simple broad classification of drinkers:

1. Irregular symptomatic excessive.
2. Habitual symptomatic excessive.
3. Addictive.[58]

Alcoholics are persons of the latter two groups. Physical complications can occur in any of the three classes, but mental complications of any type only in classes 2 and 3.

Today, among the nations of the world there are at least eight which have passed national legislation for the control of alcoholism and the treatment and rehabilitation of alcoholics. They are Sweden, Denmark, Finland, Norway, Switzerland, The Netherlands, Belgium and more recently France.[53] Of these, Sweden* has been the leader and has, since 1913, had special laws providing for the treatment of alcoholics.[23]

Repeatedly in such legislation the social nature of this illness has been emphasized.[54]

In general, the social and medical illnesses deriving from the intemperate use of alcohol have been the object of observation, study and countless efforts at control in both ancient and recent times. The accumulation of knowledge from private and public efforts has led to the present international attack on the problem—an attack that has its operations grounded in the sciences, basic and applied, physical and social.

It is obvious that alcoholism has long been viewed as both a clinical and a social issue. Karl M. Bowman, one of the pioneers in the scientific investigation of alcoholism in America, wrote in 1942: "Every medical issue is, or ought to be, a social issue, too; and some social issues, regarded strictly as such, ought to become medical issues. The latter type of social issue usually becomes suitable for effective social handling only after medicine has adopted it, but only long after. This is due to the fact that when medicine first accepts a social issue as a medical one, the social effects tend to cling to it and temporarily to becloud the purely medical integration of the problem. Medicine can eliminate these encumbrances only gradually. When this has been accomplished, however, the quondam social issue emerges as a rigid medical problem. This stage, too, is not the most productive, for at this time society does not recognize the issue as its own but tends to accept the narrow view that it belongs in the restricted sphere of the medical scientist. Finally, the medical development of the problem reaches a stage when medicine can afford to drop the role of exclusiveness with respect to it. Medical science itself now reintegrates the desirable social aspects into the problem, and it becomes manifest in its correct proportions as a medico-social issue. Society is then ready to tackle it with the greatest promise of success. This process is operative with respect to the alcohol problem."[13]

*Sweden, in its original law of 1913, defined alcoholics in terms of behavioral defects, e.g., people who in their use of alcoholic beverages were unable to care for themselves, or those who disrupted the life of a neighbor.

ALCOHOLISM IN THE UNITED STATES

From the beginning, American interest in alcoholics and in the entire problem of alcoholism has been sustained not only by voluntary efforts of individuals and organized groups, but also by the early colonial instructions in the founding charters. For example, the royal instructions to colonial Virginia of November, 1606, mandate punishment of ". . . all manner of excess through drunkenesse or otherwise, and all idle loytering and vagrant persons" as the designated governing body of the colony should see fit.[2] The varying attitudes of the more puritanical northeastern colonies are too well known to require documentation, although in recent years researchers have called attention to the fact that heavy use of alcohol was a widely accepted practice of pioneer times.

Parallel with Europe, this country entered the Eighteenth Century debate begun by Thomas Trotter as to whether habitual drunkenness was a disease or a form of moral insanity. Temperance and anti-temperance forces aligned themselves, the former reaching their greatest numerical strength in the middle of the Nineteenth Century. The Washingtonian Movement of the 1840's best exemplifies the rapid growth of the temperance movement during that period.[26]

The early suggestion (1830) of the Connecticut

Medical Society that the state set up special hospitals for alcoholics was mentioned earlier; however, it was not until 1854 that an extraordinary man, Joseph Turner, succeeded in securing the passage of "An Act to Incorporate the United States Inebriate Asylum for the Reformation of the Destitute Inebriate" by the State Legislature of New York.[34] Understandably, this precocious project was ill-fated; yet, it served to arouse public interest to the extent that within 20 years after Turner's death (1889), more than 50 hospitals for inebriates were founded in America and Europe.[35]

The temperance feeling found political expression in the National Prohibition Act of 1920, the Eighteenth Amendment to the Constitution, which, after a thirteen-year trial, was repealed in 1933 by the Twenty-first Amendment.

One of the major enlightenments to come out of this experiment was that scientific inquiry into the role of alcohol in society was the *sine qua non* for understanding, handling and preventing further extension of the problem. To this end a group of educators and scientists in 1937 formed the Research Council on Problems of Alcohol.[28]

The Research Council was incorporated in 1938 and within six months became officially associated with the American Association for the Advancement of Science. The purpose of the Council was defined as follows: "The alcohol problem has become one of the major perplexities of our civilization. On top of all the intrinsic difficulties of the situation, there have been superimposed emotional and political elements that have produced still further complications. It has become evident that nothing can be accomplished by the application of main force. If we are to find a way out, it can only be through the development of a complete factual basis on which can be built an intelligent, effective plan of action. The main and primary purpose of the Research Council on the Problems of Alcohol is to ascertain the facts; the secondary objective is to make these facts available to the public in such a way that they will do the most good."[28]

The scientific committee of the Council, under the chairmanship of Karl M. Bowman* and executive directorship of E. M. Jellinek,† immediately initiated a complete classificatory survey of world literature dealing with alcohol and inebriety. The substance of the study was published in several volumes by the Council.[13]

This work of collecting, abstracting and classifying the literature dealing with alcohol and alcoholism was taken over in 1940 by the Yale University Laboratory of Applied Physiology in New Haven, Connecticut, where the staff of the newly founded *Quarterly Journal of Studies on Alcohol* could preserve and expand it.[15]

This work resulted in the Classified Abstract Archive of the Alcohol Literature, a codified collection of some 6,000 abstracts, which, through the cooperation of WHO, has been duplicated and established in depositories in many countries§ of the world.[15]

The Yale University Laboratory of Applied Physiology, which materially aided the Council in its initial efforts, has a long and outstanding record of a multidisciplinary approach to the problem of alcoholism that began in 1930 under the direction of Howard W. Haggard. It has stimulated research on metabolic, physiological, medical, sociological and psychological aspects of alcoholism, establishing itself as a national authority on investigations relating to this problem area.

Upon the death, in 1949, of the Research Council on Problems of Alcohol—"Too much overhead, with too little money for research," said Anton J. Carlson, the council's president—the National Research Council took over the various uncompleted projects still under way.[1]

The pioneering work and continuing influence of the Yale group‡ have been, for the past 20 years, a directing force in every phase of alcoholism research, treatment and rehabilitation both nationally and internationally.

No discussion of alcoholism in the United States is complete without mention of the unique, voluntary alcoholic-treatment group, Alcoholics Anonymous, which originated in 1935.[36] This organization with 150,000 members in this country,[24] now has a counterpart in every country that is attacking the problem of alcoholism. Alcoholics Anonymous is recognized by the medical profession as a worthy adjunct to medical treatment for those alcoholics who can accept AA's traditions and credos.[6]

The acceptance of alcoholism as an illness by

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†Co-founder of the Quarterly Journal of Studies on Alcohol, official organ of Yale Laboratory of Applied Physiology—currently and since 1952 WHO Consultant, Mental Health Section, Expert Committee on Alcohol and Alcoholism.

§By 1952 depositories were designated by regional offices of WHO to the following countries: Belgium, Denmark, Finland, France, Germany, Ireland, Norway, Sweden, Switzerland, Yugoslavia, Argentina, Brazil, Canada, Chile, Uruguay, Paraguay. A number of depositories have been established in the United States by public or private agencies within the states.[16]

‡The Yale University workers have been instrumental in establishing and supporting:

1. The Quarterly Journal of Studies on Alcohol (1940).
2. Alcoholism Treatment Digest (a bimonthly publication).
3. Classified Abstract Archive of Alcohol Literature (1941).
4. Yale Clinic Plan (1944).
5. National Committee for Education on Alcoholism (independent of the Yale group since 1949) (1944).
6. Yale Center of Alcohol Studies—1945 (separate from the Laboratory of Applied Physiology).
7. Yale Summer School of Alcohol Studies (1942).
8. Summer School of Alcohol Studies in other states (1949).
9. Consultative and advisory services to all branches of federal, state and local government.

society and by organized medicine has been a slow process. Today, despite the tremendous work of the past two decades, many persons of this and other countries consider alcoholism a social blight to be handled by the correctional authorities, or, at best, to be hidden from public view by mental hospitals. In 1954, Doctor Anthony Zappala, Director of the Alcoholic Rehabilitation Division, District of Columbia Department of Public Health, wrote: "The medical definition, which is gradually finding its way into the law, describes the alcoholic as a person who has lost control of the practice of drinking alcoholic beverages to an extent that his interpersonal, family, or community relationships have become seriously threatened or disturbed.[37]

At the state level the modern attack on alcoholism began with the formal establishment of a program by Connecticut in 1945. By 1952, 38 (now 40) states and Washington, D. C., had passed legislation recognizing alcoholism as a public health problem and created boards or other official agencies to establish programs for dealing with the problem.[8] The National States' Conference on Alcoholism,* an association of administrators of state alcoholic programs, was created in 1950. Its membership comprises representatives from most states having programs for the study and treatment of alcoholism.[48]

Growing federal interest led to the following statement[9] by the Public Health Service in 1951:

"The U. S. Public Health Service's interest in alcoholism stems from its long-standing concern with the basic problems of addiction. The present consideration of alcoholism as a public health problem involves a coordinated program in which the National Institute of Mental Health serves as the focal point of the definitive approach to the problem as a whole. Two other units of the service are currently concerned with particular aspects of the problem, namely, the Division of Occupational Health of the Bureau of States' Services and the Division of Hospitals in the Bureau of Medical Services. The divisions just mentioned collaborate with the National Institute of Mental Health with respect to research and other matters of mutual interest.

"The basic public health approach to alcoholism emphasizes the need for detection and treatment of early stages, and the development of long-range programs for prevention through education and coordinated community efforts. The National Institute of Mental Health, through its community-services activities, endeavors to make clear the symptomatic nature of alcoholism, to emphasize the importance of general medical and mental health services in arresting it, to foster more adequate methods of rehabilitation, and to apply more effective measures of prevention. The approach to these objectives involves a wide base of cooperative participation of agencies working at local levels.

"The Public Health Service is prepared to support research of a basic or applied nature in the field of alcoholism. Grants for such research may be made to universities, hospitals, laboratories, other public or private institutions and to individuals in accordance with the provisions of the National Mental Health Act."

*Now the North American Association of Alcoholism Programs.

ALCOHOLISM IN CALIFORNIA

Although the law establishing California's Alcoholic Rehabilitation Commission (Chapter 22, Statutes of 1954, Extraordinary Session) was not passed until 1954, there is no shortage of historical evidence to attest the traditional interest of the State in alcoholism as both a medical and social problem. Section 3 of the five sections detailing the duties of the State's new Board of Health (now called the Department of Public Health) in 1870 reads: "It shall be the duty of the board, and they are hereby instructed, to examine into and report what, in their best judgment, is the effect of the use of intoxicating liquor, as a beverage, upon the industry, prosperity, happiness, health and lives of the citizens of the State; also what legislation, if any, is necessary in the premises."[38]

Thomas M. Logan, M.D., first executive secretary to the Board of Health, in the first Biennial Report (1870-1871) of the Board,[39] wrote of Section 3:

"In the full knowledge of the fact of the pecuniary loss to the State and to his family, by the idleness of the inebriate and the cost to the public treasury for his ultimate care and support in the induced impaired health, and in the last stage of destitution, to say nothing of the expense of measures of repression and punishment called for by breaches of the peace and crimes committed by the intemperate, we think the question is one which particularly commends itself alike to the statesman and the philanthropist.

"Without, however, attempting to arbitrate between two doctrinal extremes prevailing in regard to the dietetic usages of alcohol or vinous drinks, but simply looking at things as we find them, and as in the case of any other form of physical evil afflicting a fellow-creature, either standing by itself or implicating, at the same time, the moral and intellectual faculties, we should prefer to limit, if possible, its extent or diffusion by the encouragement of those preventive measures brought about by individual will, aided by and at the same time

aiding voluntary association with others, rather than by legislative enactment."

This comment of three-quarters of a century ago was strangely prophetic, for the entire history of activities relating to the amelioration of the alcoholism problem in this State is one of voluntary efforts. The recent creation of a State agency to deal with ". . . all phases of the treatment and rehabilitation of alcoholics . . ."[40] was a response to these long-standing, voluntary efforts combined with more recent public-group requests that the State Legislature seek an answer to the alcoholic problem.[42]

Voluntary partisan groups, as well as those interested only in treatment and rehabilitation, such as Alcoholics Anonymous, formed a very vigorous and vocal pressure to which a positive governmental response was inevitable. The traditional view of the alcoholic as a public nuisance and weak-willed wrong-doer whose activities warranted only imprisonment is gradually giving way to the modern concept of alcoholism as an illness.

In 1937, the year the Research Council on Problems of Alcohol was formed, the State of California made provision for the commitment of "dipsomaniacs, inebriates, and stimulant addicts" to State mental hospitals.[43] This action, designed to house and treat California's "problem drinkers," quickly demonstrated that at most those committed represented a very tiny portion of the alcoholics needing treatment. It was found that alcoholic admissions to mental hospitals soon comprised one-fifth of all admissions.[22] Yale estimates of the extent of California's problem in 1947 indicated one quarter of a million intemperate drinkers, and some sixty thousand chronic alcoholics (those requiring immediate remedial attention), with California rating second in the nation in per capita consumption of alcoholic beverages.[7,12] The most recent application of the Jellinek Estimation Formula[57] for the measurement of the prevalence of alcoholism in the United States indicates California as the state with the highest rate of alcoholism,[17] and San Francisco as the city with the proportionally highest number of alcoholics in the entire nation.[18]

The 1948 Emmet Daly study[5] of San Francisco's handling of the "drunk problem" served to highlight the problem and led to the establishment of the San Francisco Adult Guidance Center of the Health Department of the City and County.[14] Similarly, in 1949 the Sheriff's Office of Alameda County instituted an alcoholic-treatment clinic in the Santa Rita Rehabilitation Center at Pleasanton.[49]

The Governor's Conference on Mental Health in 1949 devoted an entire section to alcoholism and recommended the designation of an agency to carry out the responsibilities of the state in the field of alcoholism.[44]

Public health officials reemphasized the extent of alcoholism as a public health problem,[27,52] and the California Medical Association appointed a special committee to survey the problem[11] and define the role of the profession in dealing with it. In 1950 the Board of Supervisors of Los Angeles County engaged consultants from the Yale University Center on Alcohol Studies, Laboratory of Applied Physiology, to study the problem of alcoholism in Los Angeles County.[30] In 1952 the Yale workers began a study of alcoholics at the Santa Rita Rehabilitation Center, and, as in the past, a state-wide institute on the problems of alcoholism was held.[50,51]

The Weinberger Committee report and recommendations[42] on alcoholic beverage control and the 1954 Legislative Auditor's report on alcoholism[46] paved the way for the establishment of the State Alcoholic Rehabilitation Commission under Assembly Bill 9.

The Commission was authorized and directed by the Legislature to carry out the following responsibilities: (1) Investigate and study (a) all phases of the treatment and rehabilitation of alcoholics; and, (b) other factors necessary to the reduction and prevention of chronic alcoholism and other excessive uses of alcohol; (2) engage in all phases of the treatment and rehabilitation of alcoholics; and, (3) submit its first report to the Governor and the Legislature before March 1, 1957, and a final report and recommendations to the Governor and the Legislature before March 1959. The basic purposes and objectives of the Alcoholic Rehabilitation Commission were to plan, organize and establish a comprehensive program comprising three major activities: (1) Treatment and rehabilitation of alcoholics; (2) research into the causes and effects of alcoholism as well as the treatment of alcoholics; and, (3) the establishment of a state, national and international public information reservoir on the subject of alcoholism. The first act of the newly formed Commission was to call in consultants from the Yale Center of Alcohol Studies to study and to advise the Commission on a program for attack on alcoholism. In its report to the Commission, the Yale group emphasized the need for a balanced program of research, treatment, rehabilitation, and especially evaluation of existing treatment programs.[31]

The vigorous manner in which the Commission set about carrying out its responsibilities for its uniquely comprehensive program can be quickly

seen by the following table of program expenditures for the years 1954 through 1957:

***State Alcoholic Rehabilitation Commission Expenditures*[45]**
(1954-1957)

	Expended 1954-55	Expended 1955-56	Estimated Expenditures 1956-57
Administration, information and staff projects:			
Salaries	$16,614	$ 32,657	$ 53,520
Operating expense	11,052	11,316	28,913
Equipment	3,437	242	2,717
Consultants	5,683	2,323	1,000
Nature of alcoholism:			
Research projects	.....	18,000	50,000
Extent and effects of alcoholism:			
Causation survey	.....	.....	50,000
Control of alcoholism:			
Research clinic, UCLA	.....	50,000	50,000
Evaluation studies	11,583	40,000	40,000
Hospital pilot treatment	.....	.....	15,000
Community pilot clinics:			
Alameda County	.....	.....	34,000
Los Angeles	.....	.....	35,000
Sacramento	.....	.....	9,000
San Diego	.....	.....	25,000
San Francisco	.....	40,199	50,000
San Jose	.....	.....	16,000
Stockton	.....	.....	21,000
Total expenditures	$48,369	$194,737	$481,150

It will be seen that from the beginning of the Commission's program, funds were allocated and spent for evaluation studies of existing alcoholic-treatment facilities (State Department of Public Health); for augmentation of the budget of an existing alcoholic-treatment facility (San Francisco Adult Guidance Center, a voluntary outpatient clinic operated by the Health Department of the City and County of San Francisco); and for the establishment of a research clinic (University of California at Los Angeles) for study of treatment methods. Later, budgetary appropriations were made for expansions of evaluation programs; for the initiation of causation studies; for medical research into the metabolism of alcohol, the neuro-endocrine system, the relation of alcohol and the liver, precise determination of blood alcohol measurements; for studies of the impact of alcoholism on the family unit and of the effect of alcoholism on law enforcement; for a survey by the Commission executive staff as to the extent of instruction in California's public schools on alcohol and its effect on the body; and for a greatly expanded system of funds for the establishment of community pilot clinics.

Some unique features in the establishment of the community alcoholic rehabilitation clinics were that over and beyond the primary purpose of rehabilitating as many alcoholics as reasonably possible, the clinics, by contract, must carry on the following activities: (1) Evaluation of patient status (diagnosis); (2) treatment and rehabilitation; (3) information—public and professional; (4) community organization and coordination; and, most importantly (5) follow-up (evaluation of the treatment program). A full account of the Commission's activities for the period cited is given in the 1957 Interim Report to the State Legislature.[45]

In the 1957 session of the State Legislature, a new alcoholic rehabilitation law was passed which abolished the Commission and created within the State Department of Public Health an Alcoholic Rehabilitation Division.[41] The responsibilities of the Health Department, like those of its predecessor, include treatment, rehabilitation and investigation.

The placement of a total alcoholic rehabilitation program in the Health Department indicates the state's recognition of alcoholism as a public health problem and responsibility. This action insures continuing support for treatment, rehabilitation and investigation with prevention as the ultimate goal.

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Gallstone Intestinal Obstruction

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• The literature on the subject and the records of 40 cases of proven gallstone obstruction in the small intestine observed at the Los Angeles County General Hospital over a 27-year period were reviewed.

The incidence of this type of obstruction is about 1.5 per cent of all cases of mechanical intestinal obstruction; and it occurs more often in women than in men—the ratio was 3.7 to 1 in the Los Angeles County General Hospital series.

In general, the majority of patients are in the seventh decade of life, although in the present series the age average was well over 70 years.

Gallstones large enough to cause intestinal obstruction almost invariably reach the intestinal tract through a fistula between the gallbladder and the duodenum.

The symptoms of gallstone obstruction are principally those of mechanical obstruction of the small bowel. The usual site of obstruction is the distal ileum. When gallstones are the cause of obstruction, the symptoms may be intermittent.

Surgical operation is the treatment of choice. Exploration should include a complete examination of the intestinal tract to make certain multiple stones are not overlooked, and the right upper quadrant should be palpated for the presence of an acutely inflamed gallbladder or more calculi.

INTESTINAL OBSTRUCTION from gallstones is associated with one of the highest mortality rates among the various kinds of intestinal obstruction. While it is important to recognize as quickly as possible the specific nature of any obstructing lesion, this is particularly true of obstruction of obturator type due to enteric gallstones. Operation is the treatment of choice. Because the symptoms caused by the migration of a calculus from the biliary tract into the intestines are likely to be intermittent and none too characteristic, diagnosis is frequently delayed and often impossible.

A review of the subject and a brief summary of 40 cases of intestinal obstruction due to gallstones, as observed in 38 patients at the Los Angeles County General Hospital since 1930, will be presented.

HISTORY

Bartholin,[1] in 1654, was the first to describe the erosion of a gallstone into the intestinal tract. Courvoisier[2] made a survey of the literature in 1890 and analyzed 131 cases; and in 1914 Wagner[10] published an analysis of 340 cases that remains as one of the most thorough surveys of the literature on this subject. Reports of approximately 800 cases of gallstone obstruction have appeared in medical records to this time.

INCIDENCE

The incidence of intestinal obstruction of this type is difficult to state. The average incidence reported in many series covering a total of 30,508 cases of intestinal obstruction of all kinds was 1.5 per cent.

In the 27 years 1930 through 1956 there were 6,063 cases of small bowel obstruction observed on the surgical wards at the Los Angeles County General Hospital. The records of 40 proven cases of gallstone intestinal obstruction in that period were available—an incidence of 0.6 per cent.

In recent years at this hospital there has been an increasing awareness of obstruction of this type. Case recording has improved. Although only 22 cases of gallstone obstruction were noted from 1930 to 1950, in the six-year period 1951 through 1956 there were 18 such cases among a total of 1,697 cases of small bowel obstruction, an incidence of 1 per cent.

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Gallbladder disease occurs from two to three times as often in women as in men.[3,8] About the same ratio holds with regard to obstructions due to gallstones. In the earlier reports in the literature the female-to-male ratio of gallstone obstruction was similar to that for the sex incidence of cholelithiasis. In the more recent literature there is an increasing preponderance of females with gallstone obstruction. In the Los Angeles County General Hospital series the ratio was 3.7:1 (Table 1).

The average age of patients with gallstone obstruction has changed somewhat from that reported in earlier years. In Courvoisier's review published in 1890 the average age of female patients was 58 years and of males 60. More than 80 communications were reviewed in surveying the reported incidence of gallstone obstruction in more recent times.

TABLE 1.—*Incidence of Gallstone Intestinal Obstruction by Sexes*

	Females	Males	Ratio
Courvoisier[2]-Wagner[10] (to 1914)	195	80	2.4:1
Collected reports (1914-1956)	249	33	7.5:1
Present series (1930-1956)	30	8	3.7:1

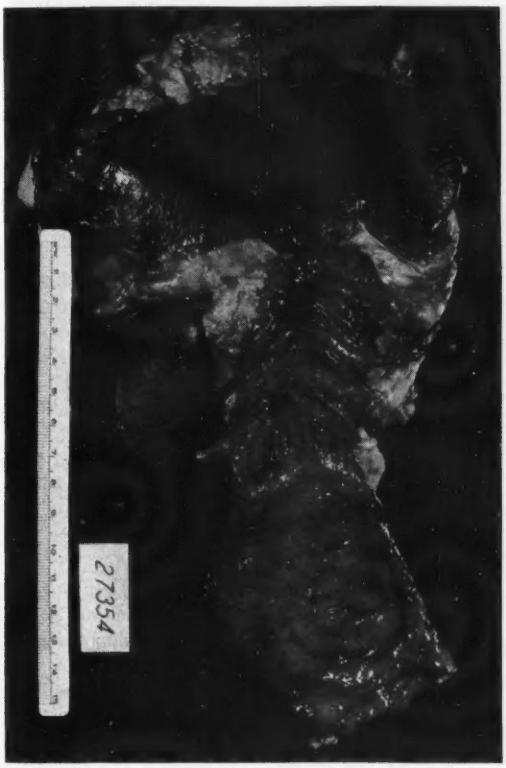

Figure 1.—A typical cholecystoduodenal fistula in autopsy specimen from case of gallstone intestinal obstruction. The large obstructing calculi can also be noted.

Although not a complete survey, this covered a combined group of more than 400 cases. The average age of patients was 64 years and was about the same for men as for women.

As is usual for a series of cases reported from the Los Angeles County General Hospital, the average age of the patients was greater than for other comparable series—for women, 71 years; for men, 76. The range was from 42 to 90 years.

The youngest patients of record were a 25-year-old woman reported upon by Wortmann[11] and a 26-year-old man whose case was reported by Ritter.[7] The 90-year-old woman in the present series and the 93-year-old woman whose case was reported by Shore, Jacob and Cannon[9] are the oldest.

PATHOGENESIS

Gallstones may enter the intestinal tract by eroding through some part of the gallbladder or common duct. It is estimated that to cause an obstruction a calculus must be more than 2 cm. in diameter. In the great majority of instances the stones causing obstruction enter the bowel through a cholecystoduodenal fistula such as the one shown in Figure 1. In all cases in the present series entry was through a fistula of this type.

Other modes of entry are much less common. Fistulae have been reported connecting gallbladder to jejunum, ileum, stomach, colon, renal pelvis, the urinary bladder and even the vagina and bronchus. The reported incidence of spontaneous internal biliary tract fistula as observed in biliary operations, ranges from 1 to 4 per cent.[4,5]

Occasionally a large calculus may erode partly through the gallbladder or common duct into the duodenum. Symptoms of pyloric obstruction may result.

The process of internal fistula formation begins with recurrent cholecystitis. A stone then causes ulceration and erosion of the gallbladder mucosa by pressure on the softened, inflamed gallbladder wall. In response to this process, pericholecystitis is produced and adjacent viscera become adherent to wall off the impending perforation. A fistula is finally formed between the gallbladder and the adjacent viscus, which is usually the duodenum. The gallbladder, once free of calculi, rapidly becomes a blind sinus tract and contracts down to a small fibrous remnant. This pathologic sequence was confirmed in the study of the cases in the present series.

SYMPTOMS AND DIAGNOSIS

Often clinical evidence of fistula formation and passage of the stone into the intestinal tract is scanty. When a stone does pass through a fistula, there may be severe pain and nausea and vomiting, or the calculus may enter the bowel with only minor effects. Regardless of the sequence of onset, the paramount symptoms are those of an intestinal obstruction. The train of symptoms that ensues depends principally on the size of the stones. In general, large stones will cause more persistent symptoms by obstructing early. Smaller stones may pass down and out of the alimentary tract without causing severe symptoms. If the stone is irregular or rough, it may produce intense local trauma with subsequent spasm. Characteristically, gallbladder obstruction is less clear-cut than any other kind of mechanical obstruction.

Not infrequently the symptoms will be those of intermittent obstruction, the nausea, vomiting and colic subsiding completely as the stone becomes dislodged temporarily and moves on down the bowel. Five of the cases in the present series were of that nature; all but one were misdiagnosed.

Constant pain, which may be localized, is consistent with the impaction of the stone at one point in the intestine and severe local irritation, edema and, not infrequently, gangrene. In five of 37 cases in this series in which operation was done, gangrenous areas of various sizes were present.

Figure 2.—A flat film of the abdomen showing classical findings of gallstone intestinal obstruction—dilated loops of small bowel, air in the biliary area, and radiopaque calculi in the lower abdomen.

In general, about 80 per cent of the patients have a history of biliary tract disease or of symptoms compatible with that diagnosis. Of the 38 patients in the present series, 19 gave either a history of proven gallbladder disease or of symptoms adequate to make a presumptive diagnosis. In 11 others the history was entirely negative for symptoms referable to the biliary system. Of the remaining eight, four were too ill to give good histories, and in the others the history as recorded was inadequate in detail.

Rigler, Borman and Noble[6] set down radiologic criteria which help in making an accurate diagnosis of this disease by x-ray examination of the abdomen. They listed four roentgen signs that are indicative of obstructions due to gallstones: (1) Air or occasionally contrast medium in the biliary tree; (2) direct visualization of the stone or sometimes indirect visualization of the stone by means of contrast medium within the intestine; (3) a change in position of a previously noted stone; and (4) in all of the foregoing situations, roentgen evidence of complete or partial obstruction.

The most frequently observed diagnostic sign is air in the biliary tract. This enters from the intestinal tract through the fistula produced by the migrating stone. Films can be centered over the right upper quadrant. A larger x-ray film should be searched carefully for radiopaque stones. They are difficult to see when overlaid by intestinal loops filled with fluid and gas or when they are superimposed on skeletal shadows. Figure 2 illustrates two positive roentgen signs often seen in gallstone intestinal obstruction—radiopaque stones in the pelvic area (two stones in this case) as well as air in the biliary tract. In this series the majority of abdominal films lacked the positive diagnostic signs. In only one instance was air seen in the biliary system. In six cases the stone was visible among dilated intestinal loops. In another case, an earlier film was available which showed a change of position of a radiopaque stone from the right upper quadrant to the left lower quadrant.

TABLE 2.—Site of Gallstone Intestinal Obstruction

	Courvoisier[2]	Wagner[10]	Present Series
Duodenum	3	9	....
Jejunum	5	30	3
Upper and mid-ileum	23	103	9
Lower ileum	10	53	26
Ileocecal valve	7	15	1
Appendix	2	....	....
Sigmoid colon	2	13	1

In the total series the operating surgeon properly diagnosed gallstone obstruction preoperatively in 17 instances. In 16 other cases the diagnosis was mechanical intestinal obstruction. No diagnosis was made in two instances; neither patient was operated upon and both died. A third case was misdiagnosed as a sealed off perforated viscus; operation was not done and the patient died. In the remaining cases the diagnoses were acute appendicitis, mesenteric vascular occlusion and carcinoma of the cecum. The correct diagnosis in these cases was made at operation and the obstructing calculi were removed.

SITE OF OBSTRUCTION

The ileum is the site of impaction and obturation obstruction in the majority of cases of gallstone obstruction. A comparison of the sites of obstruction in this series with the collected series reviewed by Wagner and Courvoisier is made in Table 2.

Four patients in this series had multiple stones. One patient had two large stones impacted in the terminal ileum and two other large stones in the common duct. In another case obstruction 12 inches from the ileocecal valve was due to an impacted stone 3.0 cm. in diameter, while four smaller ones, all less than 1.0 cm., were noted to have caused erosion with perforation in the mid-ileum above the site of impaction. In the same case another large stone was removed from the gallbladder at operation. In another case two large calculi were removed from the terminal ileum. One patient had nine stones in the mid-ileum.

Two patients in the series were treated for recurrent episodes of gallstone intestinal obstruction. In

TABLE 3.—*Mortality Rates Associated with Gallstone Intestinal Obstruction*

	Mortality Rate (Per Cent)
Collected series:	
700 cases	50.0
Present series (1930-1957):	
40 cases	40.0
Collected series (1950-1956):	
103 cases	18.4
Present series (1951-1957):	
17 cases	24.0

one case the second obstruction occurred approximately two weeks after operation and in the other the interval was two months.

TREATMENT AND OPERATIVE TECHNIQUES

Early operation is the treatment for gallstone obstruction. Time must be taken to evaluate and partially correct electrolyte and fluid losses, since many of the patients enter the hospital late in the course of the disease. At exploration the entire intestinal tract must be examined for multiple calculi. Occasionally the stone may be pushed into the colon and around into the rectum. This is the procedure of choice if it can be done easily; however, these patients are usually elderly and frequently in desperate condition and any excessive trauma must be avoided. If the stone can be loosened and removed proximally, it can be removed through a segment of intestine where there is less edema and inflammation. The enterotomy incision should be closed transversely. A 1 per cent solution of Neomycin into an isolated segment containing the stone is a procedure of value. During the operation the right upper quadrant area should be examined to ascertain the type of biliary disease present. If additional large stones are present they should be removed by cholecystostomy if the patient's condition is not too precarious.

RESULTS OF TREATMENT; MORTALITY (Table 3)

In general the longer the duration of symptoms before operation, the higher the mortality rate. In some cases obstruction is intermittently relieved as the stone shifts position. Nevertheless, delay in diagnosis is the chief reason for the high mortality in this type of intestinal obstruction. Sixteen of 38 patients in the present series died. In two cases cholecystostomy was done concomitantly with release of the obstruction, and in one case cholecystectomy. One elective cholecystectomy and two cholecystostomies were done months after the acute obstruction for relief of persistent biliary symptoms.

The average of mortality rates reported in various collected series was 50 per cent (Table 3). At the Los Angeles County General Hospital there were 24 recoveries and 16 deaths in the 27-year period surveyed. Three of the deaths were nonoperative. This was an overall mortality of 40 per cent and an operative mortality of 32.5 per cent. In more recent series the mortality rate has been increasingly better, although it still remains quite high when compared with the rates associated with small bowel obstruction of other types.

When the advanced age of the Los Angeles County General Hospital patients is considered, it is remarkable that the mortality rate compares so favorably with the general average. In spite of better diagnosis and undoubtedly improved facilities for supportive care the gross mortality rate for the 17 patients treated in the last six years of the study was still 24 per cent and the operative mortality rate 18 per cent.

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Absence from School

Its Relationship to Illness in the Community

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• **The absences at one elementary school in Los Angeles and the reasons for the absences during the school year 1954-55 were studied. The absence rate was then compared with the rates during two previous years at the same school and with the absences at three other elementary schools in Los Angeles and with one industry. There were similar absence curves for the several years in the single school and among the schools and industries which were compared. The highest absence rates were found during the winter months and common respiratory tract disease accounted for the highest percentage of absences from school throughout the year.**

ABSENCE FROM SCHOOL due to illness is a major public health problem from the standpoint of epidemiology and health education. As progress is made in medical knowledge, skill and armamentarium, some diseases are brought under control or are virtually eliminated, while others come into sharper focus. For example, smallpox is now practically nonexistent in California, but the common respiratory tract infections are of increasing importance and interest to us as more is learned about the nature of viral diseases. It is important, therefore, to recurrently review the causes of absence from school in various areas and also to attempt to correlate the causes with the frequency of illness in the community. For it is important to know whether the absences due to illness from a given school are peculiar to that school or are only a reflection of prevalent illness in the community. Physicians, school authorities and parents desire such information regarding the causes of absence from school to guide them in establishing effective policies and attitudes.

This study is concerned with establishing the pattern of the absences from school in the University Nursery and Elementary School of the University of California at Los Angeles. These patterns are compared with the absence patterns of other years in the same school, with patterns in other schools in diverse areas of Los Angeles in the same year, and with patterns in one major industry, a department store. Data has also been obtained in as much detail as possible regarding the reported causes of absence from the University school during one year.

MATERIALS AND METHODS

The University Nursery and Elementary Schools are on the campus of the University of California at Los Angeles. The enrollment of 372 children is divided in the following way: (1) Nursery school containing three sections of ten to fifteen children each, (2) kindergarten with two sections of twenty children each, and (3) grades 1 to 6 with two classes at each grade level having twenty-five students each. The children who attend are mainly from middle and upper class families. They are well cared for at home and all have private medical supervision.

During the school year 1954-1955 accurate records were kept of every absence from school and of the major reason for this absence as stated by the parent. The information was entered on a three by five inch printed card (shown below) and this, instead of the usual absence note, was brought to the teacher when the child returned to school after an absence. For the preceding school years 1952-1953 and 1953-1954, accurate absence data was available, but the causes for absences were not documented.

The data obtained from the three other schools representing different health districts and from the one industry, a large department store with 1,350

UNIVERSITY OF CALIFORNIA, LOS ANGELES
University Elementary School

Name:..... Grade:..... Date.....

ABSENCE FROM SCHOOL WAS DUE TO

..... Respiratory infection (cold, cough, sore throat, earache, flu, laryngitis) Fever—Yes..... No.....
..... Intestinal upset (vomiting, diarrhea)
..... Measles—3-day..... or Regular.....
..... Mumps
..... Chickenpox
..... Allergy (specify).....
..... Other (holidays, illness).....

Dates absent..... Signature.....

From the Department of Pediatrics, School of Medicine, University of California at Los Angeles, Los Angeles 24.

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Submitted October 11, 1957.

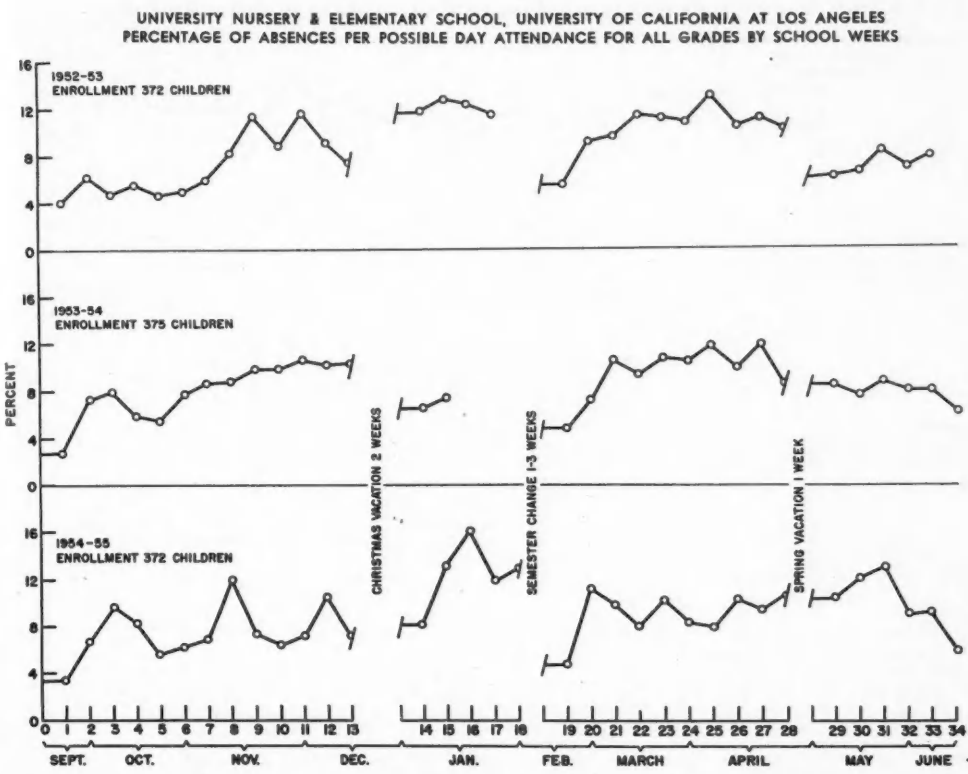
UNIVERSITY NURSERY & ELEMENTARY SCHOOL, UNIVERSITY OF CALIFORNIA AT LOS ANGELES
PERCENTAGE OF ABSENCES PER POSSIBLE DAY ATTENDANCE FOR ALL GRADES BY SCHOOL WEEKS
1952-53
ENROLLMENT 372 CHILDREN
1953-54
ENROLLMENT 375 CHILDREN
1954-55
ENROLLMENT 372 CHILDREN
PERCENT
CHRISTMAS VACATION 2 WEEKS
SEMESTER CHANGE 1-3 WEEKS
SPRING VACATION 1 WEEK
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 —WEEKS
SEPT. OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY JUNE —MONTHS

Chart 1.—A comparison of the absences from the University Nursery and Elementary School of the University of California at Los Angeles for the school years 1952-53, 1953-54 and 1954-55. The absences are expressed as percentage of possible days attendance.

TABLE 1.—*A Table of the Causes of Absence from the University Nursery and Elementary School of the University of California at Los Angeles for 1954-55 Given as Percentages of the Total Number of Absences for Each Month.*

	Respiratory Infection	Intestinal Upset	Measles	Mumps	Chickenpox	Allergies	Other Illness	Skin Infection	Accident and Injury	Family and Personal*
September	53.1	3.1	0	0	0	1.1	4.2	2.1	0	36.4
October	59.9	5.6	0	0.3	0	1.6	8.7	0	0.6	23.3
November	62.1	8.8	0	0.5	0	1.4	9.2	0.5	0.5	17.0
December	65.1	10.7	0	0	0	1.7	9.0	1.3	0	12.2
January	71.1	10.5	0	0.5	0	0.8	6.6	0.8	0	9.7
February	64.0	19	0.4	0.4	0	0.4	3.3	0.8	0.4	11.3
March	68.4	11.1	1.0	0	1.4	0.3	4.6	1.0	0	12.2
April	53.2	7.8	0.8	0	7.4	1.2	3.3	0.8	0.4	25.1
May	41.8	7.9	3.7	1.8	9.3	0.5	9.3	1.9	1.4	22.4
June	43.0	2.4	20.8	0.6	2.4	1.8	3.2	0	1.2	24.5

*Included household moving, trips, emotional upsets, appointments with physicians.

employees, was gathered by the Los Angeles City Health Department during 1954-1955 in an attempt to follow community illness trends.

From the raw data obtained from these surveys, the percentage of absence from school or work per possible days of attendance was computed by the following formulation: Total number of days of absence in the school or industry, divided by the number of persons times the number of days of school or work during the week, times 100.

Thus the findings for the University Elementary School for the three years could be compared, as could the absence data for the University Elementary School and one local industry. Furthermore, by use of the material obtained from the absence cards, a table was constructed listing causes of absences

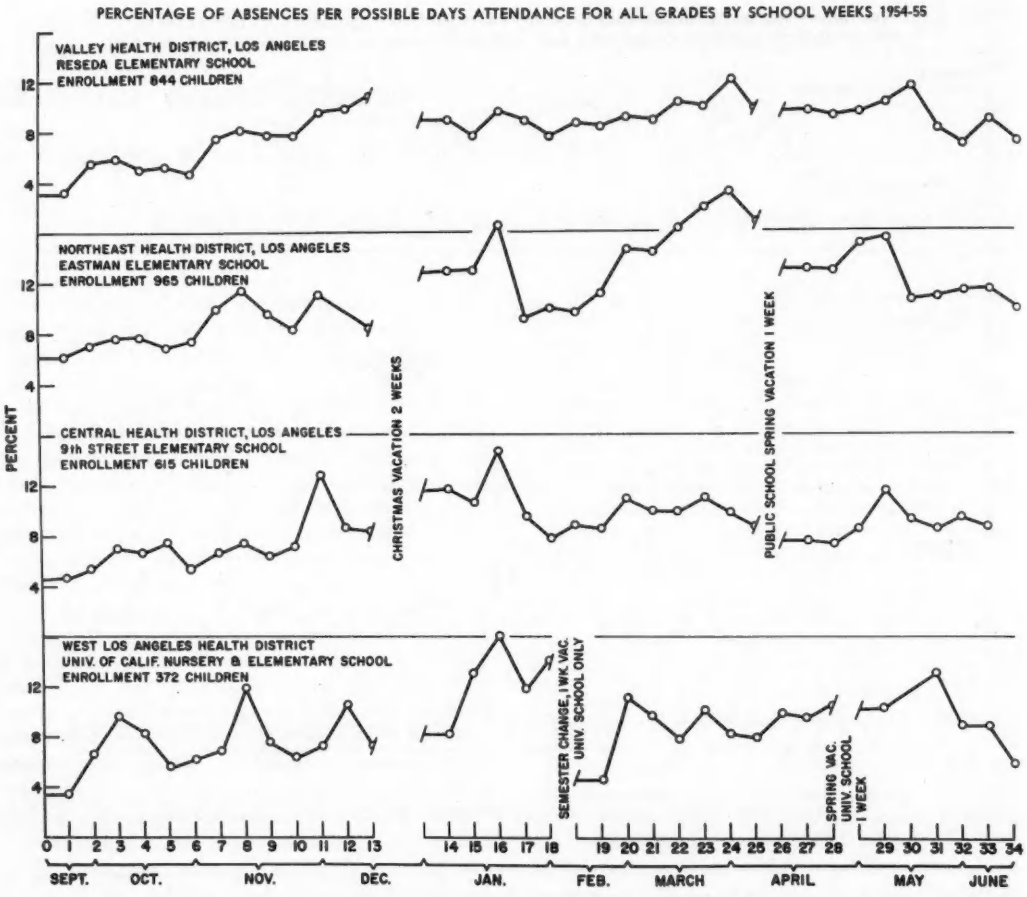

Chart 2.—A comparison of the absences from elementary schools in four separate health districts in Los Angeles during the school year 1954-55. These absences are expressed as per cent of possible days attendance.

by months and showing what proportion of total absences was ascribable to each cause.

The absence data from the University School for the years 1952-1953, 1953-1954 and 1954-1955 was related, in a graphic chart, to the number of possible days of attendance (Chart 1). In these graphs a tendency for the proportion of absences to increase through November and December, to decrease in late January or early February and then to increase again until late April or May was illustrated. That the sharp drop in late January or early February is not necessarily related to the vacation period will be discussed later. The high proportion of absences in May of 1955 is related to a high incidence of measles and chickenpox at that time. It is interesting that each year, except 1952-53, within the first two to four weeks after the beginning of school there is a small but definite rise in the number of absences. It is significant, however, that although after some of the brief vacations during the school year the absences decreased, after others they were the same or increased. The average proportion of absence per possible days' attendance for the three years was very similar—8.5 per cent, 8.4 per cent and 8.9 per cent for the three school years 1952-53, 1953-54 and 1954-55.

The data on absences for the school year 1954-55 for the four elementary schools in the separate districts in Los Angeles including the University School were graphed together (Chart 2). The four schools show trends for greater numbers of absences during the winter months as compared with the fall. Some of the peak periods of absences are in identical weeks in at least three of the schools, particularly in December and January. The relatively large number of absences in all of the schools during the late spring months, April and May of 1955 probably was related to an unusually high incidence of measles in Los Angeles in that year. This is also

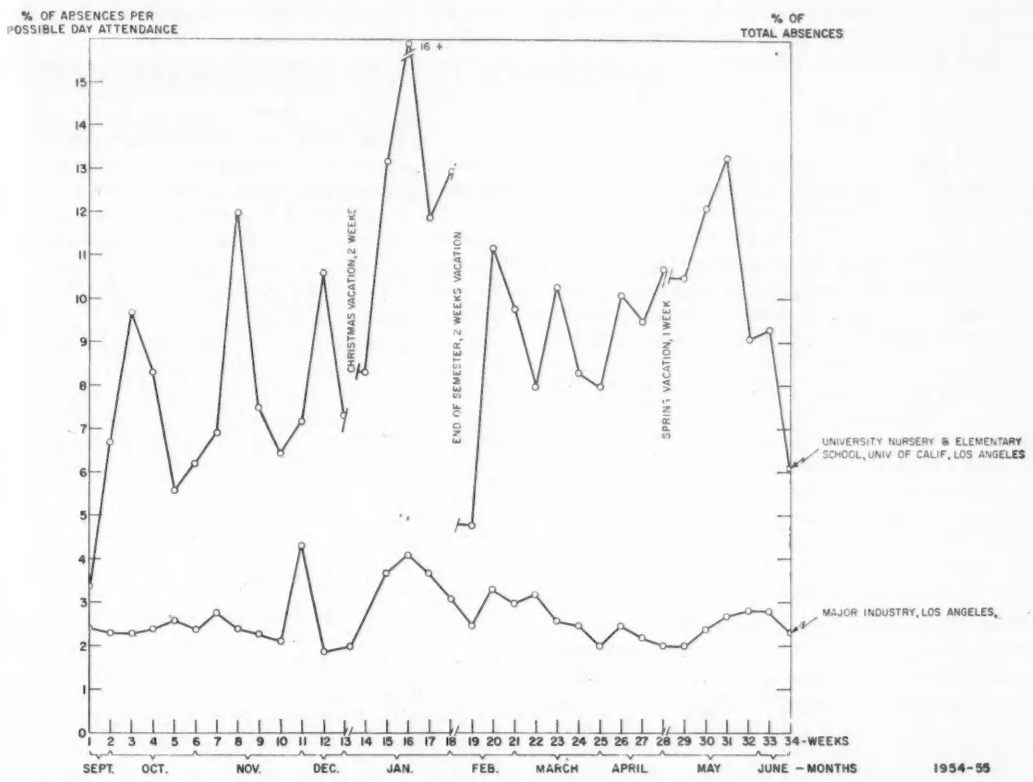
% OF ABSENCES PER POSSIBLE DAY ATTENDANCE
% OF TOTAL ABSENCES
16 +
CHRISTMAS VACATION, 2 WEEKS
END OF SEMESTER, 2 WEEKS VACATION
SPRING VACATION, 1 WEEK
UNIVERSITY NURSERY & ELEMENTARY SCHOOL, UNIV OF CALIF, LOS ANGELES
MAJOR INDUSTRY, LOS ANGELES
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34-WEEKS
SEPT. OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY JUNE — MONTHS
1954-55

Chart 3.—A comparison of absences from the University Nursery and Elementary School of the University of California at Los Angeles and the absences from a major industry in Los Angeles (a department store) during the school year 1954-55.

shown in Table 1. The average proportions of absences per possible days' attendance for each school for the entire year 1954-55 were: Reseda, 8.2 per cent; Eastman, 11.5 per cent; Ninth Street, 9.0 per cent; University Elementary School, 8.9 per cent.

The absence data for the University Elementary School and one major industry were graphed together, allowing more space for each point plotted than on the previous graphs (Chart 3). This shows the rapid rise and fall of the absences in the elementary school compared to the rather stable attendance in the industry. However, in the early part of December and in the middle of January and to a lesser extent in February and March, peak absences occurred in the industry and coincided with peaks in the elementary school. It is interesting that there was a sharp decline in the absences after the between-semesters vacation in the elementary school in January. However, there was a similar decline in the absences from the industry during this same period and the industrial employees did not have a vacation period. This would suggest that the incidence of illness in the entire community was declining at that time.

In Table 1 are indicated the causes of the absences from the University Elementary School in 1954-55 by per cent for each month. In every month respiratory tract diseases were the major cause of absence; they were blamed for more than 60 per cent of the absences from November through March. The incidence of intestinal upsets was increased during the same period as that in which respiratory infections were increased. Mumps seemed to occur sporadically throughout the year while measles and chickenpox came in the late spring. Absences for allergic diseases did not follow any particular pattern. Other causes of absence because of illness included infectious mononucleosis, infectious hepatitis, undiagnosed fevers and surgical operation. In these groups absences did not seem to follow any temporal pattern, nor did absences because of skin infections. It is interesting that accidents and injuries are minor causes of absence from school. As to the absences for family and personal reasons, the only explanation that comes to mind for the fluctuations

in incidence of absence of this order is that possibly the September high incidence may reflect delays in returning from summer vacations.

DISCUSSION

In this study it was observed that in the University Nursery and Elementary School the periods of peak incidence of absences were fairly similar from year to year. Furthermore, in 1954-55 the periods of increased absences in other schools in divers areas of Los Angeles and in one major industry (department store), were similar. An analysis of the reported causes of the absences for the year 1954-55 clearly indicated that common respiratory tract disease was the dominant cause of absences throughout the year, but especially so in the winter months when the frequency of absences was the highest. That the common respiratory diseases are generally the commonest cause of absences from school has been amply demonstrated in many areas.[2,4,6,7,8] Furthermore, Dingle[3] and Lidwell[5] demonstrated that common respiratory tract disease is the major cause of illness in the community.

It is clear, then, that in the problem of school absence the problem is not primarily epidemic childhood diseases, such as measles, mumps and chickenpox or such serious problems of the past as diphtheria and smallpox; the problem in the schools is common respiratory tract disease, as it is in the community as whole. Furthermore, respiratory tract disease increases in frequency in the school and in the community at the same time, as was noted in this study and amply illustrated by Dingle[3] and Lidwell.[5] It is interesting that both of these investigators indicated that in families with school children there is a greater than ordinary incidence of common respiratory disease in the parents and in pre-school siblings. It is possible that the school may play a significant role in the dissemination of the illness. It is interesting in this regard that a small but sharp peak of absences was noted about the second to fourth week after school starts in the fall. Dingle, in his study of Cleveland families, indicated a similar peak in respiratory infections in the month of September, but did not comment on any relationship with the opening of school. It would seem that the process of bringing all the children together in the school may be sufficient to initiate a sudden rise in the common respiratory diseases.

It is felt that the common practice of excluding the pupil from school after symptoms of upper respiratory infection develop may not be very effective. When the incidence of common respiratory infection is high in the community and in the school, many children attend whose symptoms are so minimal they go unnoticed by parents, teachers and the child. Furthermore, Andrewes[1] demonstrated in experimental subjects that respiratory disease can be transmitted from one person to another the day before symptoms develop.

It is important, therefore, that as much information as possible be gathered regarding the spread of common respiratory tract diseases in the school and community, and regarding individual resistance to infection, so that ultimately sound school health policies may be developed and health education be more realistic.

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No. 1 Killer

Automobile accidents are responsible for half the peacetime deaths in the Army.

Prepartum Nausea and Vomiting

A New Approach to Treatment

WILLIAM G. CALDWELL, *M.D.*, and KENNETH HOBBS, *M.D.*, Los Angeles

• **A controlled study of 101 patients indicated that a combination of amphetamine and Rauwolfia was effective in the treatment of prepartum nausea and vomiting. Good to excellent results were obtained in 53 (83 per cent) of 64 patients who received the combination. Five patients reported fair results; six were not benefited by the treatment.**

Only five of a control group of 37 patients who received placebos reported good results.

In addition to relief of the symptoms of nausea and vomiting, concomitant emotional disturbances, notably anxiety and depression, were alleviated by the drug combination.

THE IMPORTANCE of an adequate therapeutic approach to the control of nausea and vomiting of pregnancy is sometimes overlooked. Yet, when we consider that these symptoms are a complication in more than half of all pregnancies—one observer said they occur in nearly 80 per cent[2]—it is evident that they represent a very real problem. Many women, moreover, have moderate to severe symptoms throughout the greater part of the gestation period. Any disorder with so high an incidence cannot be ignored, and promising therapeutic measures of control merit careful investigation.

The lack of definitive etiologic knowledge of the emesis of pregnancy makes it difficult to evaluate either the need for or the efficacy of drugs. There are a number of theories ascribing an organic basis for prepartum nausea and vomiting, and endocrine, allergenic and nutritional factors may all play a role in this syndrome. But whatever the physiological predisposition, there is little doubt that psychological factors are a determinant in both mild and severe nausea and vomiting of pregnancy, and the extent and degree of these symptoms have been shown to correlate with the emotional stress to which the patients were subjected before and during the pregnancy.[5]

In a previous study[1] it was demonstrated that alleviation of emotional symptoms by the administration of an antidepressant drug was highly effective in the treatment of prepartum nausea and vomiting. The results obtained, moreover, gave added support to the theory that the syndrome is psychic in origin. The present study was undertaken to evaluate the efficacy of a combination of two mood-elevating drugs, and to confirm the value of such a therapeutic approach.

METHOD

The present study, conducted over a 16-month period, included a total of 101 pregnant women. They varied in age from 13 to 38 years, more than half being in the 14 to 20 year range; and the group included members of the White, Negro and Mexican races. All patients complained of persistent nausea and/or vomiting, and symptoms were of such severity that the women themselves requested treatment. Each was an unwed mother, and was an outpatient at St. Anne's Maternity Hospital, in Los Angeles, a specialized teaching and charitable institution devoted to caring for unwed mothers. In addition to the general psychic unrest produced by pregnancy, these patients exhibited an unusually high degree of tension, anxiety and depression as a result of the stress of self-recrimination and social condemnation. Overweight was a frequent complaint. This symptom is encountered frequently in unwed girls and is usually associated with some form of depression or nervousness.

The medication selected for clinical trial contained in a single tablet* 1 mg. of the alseroxylon fraction† of rauwolfia serpentina and 5 mg. of amphetamine sulfate. The efficacy of this combination as a mood-elevating agent when symptomatic complaints are exaggerated by underlying psychogenic factors, its anorexic effect in controlling the appetite, and freedom from side effects had been well established.[3,4,6]

The prescription that each patient started with was one tablet three times daily half an hour before meals. Dosage was later adjusted to the individual, some patients finding that two tablets per day were adequate for maintenance of effect. A number of patients were used as controls and received placebos instead of medication. The double-blind technique was followed in the dispensing of the tablets; neither

From St. Anne's Maternity Hospital, Los Angeles.
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*Rauwidrine®—Riker Laboratories, Inc., Los Angeles.
†Rauwiloid®—Riker Laboratories, Inc., Los Angeles.

patients nor medical personnel knew which preparation was being administered.

RESULTS

At the termination of the study it was found that 64 of the patients had received active medication and 37 had been given placebos. Specially designed rating charts had been distributed to the women so that they could keep a record of the effects obtained while participating in this trial. Of the 64 who received the antidepressant drug combination, 53 (83 per cent) reported good or excellent results. Only five of the 37 women given placebos reported similar results. The results, in more detail, were as follows:

	Excellent	Good	Fair	None	
Rauwidrine	30	23	5	6	64
Placebo	1	4	8	24	37
					101

It is of further interest that four patients who were receiving placebos and obtained no relief of symptoms after several weeks, requested other medication and were given another envelope of unidentified tablets. All reported good or excellent effects with these, and subsequent identification showed that in all four cases the tablets contained the drug combination. Although it is probable that some of the patients who received active medication obtained a "placebo response," it is felt that the results of this trial are significant in indicating a specific therapeutic action for the drug combination.

In patients receiving benefit from the medication, there was usually complete alleviation of symptoms by the second week of therapy. Concomitant with the disappearance of nausea and vomiting, the patients experienced a sense of well-being with relief of anxiety and depression. They were less nervous and did not give way to "crying spells." Appetite control, with desirable loss of weight or maintenance of weight was a further benefit of therapy for many of the women.

Of particular importance was the almost complete lack of side effects in this group of patients. Only three reported wakefulness while taking three tablets per day. This was overcome by reducing the dosage to two tablets daily. One patient reported the medication made her ill, but was unable to clarify her statement in any way. No other untoward effects were noted. In the group who received only placebo, three patients felt that therapy produced increased emesis. It should also be noted here that the drug combination produced no adverse effect on fetus or mother; babies were normal in every respect and there were no complications in delivery attributable to the medication.

COMMENT

It is felt that the results of this study clearly illustrate the effectiveness of an antidepressant medication, and the value of such a therapeutic approach, for pregnant patients with nausea and vomiting caused or aggravated by anxiety and depression. This work confirms the senior author's report[1] that amphetamine is probably the agent of choice in these cases, and demonstrates certain advantages of the Rauwolfia-amphetamine combination over the latter drug alone. The almost complete absence of jitteriness and insomnia due to amphetamine was noticeable. This is in agreement with other reports[4,6] that the combination offers additive mood improvement with elimination of any side reactions produced by sympatheticomimetic drugs. We have noted that often the use of barbiturates is necessary in dealing with excessively agitated patients, and were pleased therefore to discover that the use of this drug combination minimizes the necessity for additional barbiturate therapy, an observation also reported by Prouet.[4]

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Bursitis—Often an Inexact Diagnosis

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• **The word *bursitis* is like *arthritis* in that it must be modified by another before it has meaning precise enough for useful medical description and communication. It is used loosely and generally to describe a number of clinical and pathological entities, among which are true primary bursitis, cystic degenerations, calcareous deposits in ligaments and tendons, and so-called "chronic sprains" where a calcified deposit cannot be demonstrated.**

It is possible to differentiate between these four entities in the light of present-day knowledge and to make a more accurate statement of diagnosis.

Bursitis is often a symptom . . . not a primary disease and hence not a proper diagnosis.

Calcareous deposits in ligaments and tendons with evident inflammatory reaction have been described in many locations. No area seems to be immune but the rotator cuff and supraspinatus tendon of the shoulder show the greatest incidence. The term *inflammatory tendinitis* appears to be a reasonably useful one for describing the clinical manifestations of the disease. For further accuracy it can be modified by saying that it is with calcification or without calcification as the case may be.

There might be serious consequences from confusing the pain radiation of inflammatory tendinitis (with or without calcification) around the trochanteric region of the hip with true sciatic neuritis arising from a herniated intervertebral disk.

THE TERM *bursitis*, although listed as a diagnosis, still comprises a number of clinical and pathological entities which can and should be separated. Not only that, to use the term *bursitis* often violates good medical practice by naming the symptom as a diagnosis. The fuzzy thinking that this engenders tends to hinder the development of knowledge of an interesting group of diverse pathological conditions, many of them rewardingly responsive to treatment.

Nothing in this paper is new. All of the information contained in it has been reported on before. The purpose of this communication is to help direct the use of the available knowledge in a way to make more accurate diagnoses possible.

Bursitis is used as a diagnosis loosely and generally to describe a number of clinical and pathological entities, among them primary bursitis, cystic degeneration, calcareous deposits in ligaments and tendons and so-called "chronic sprains" where no calcified deposit can be demonstrated.

Primary Bursitis

Primary bursitis can be separated readily from the others by definition: It means primary inflammation of a bursa. If it is secondary to another process, then by definition it is a symptom and not a diagnosis.

Primary bursitis is commonly and best seen in the olecranon or prepatellar areas where these more superficially placed synovial sacs become directly irritated from multiple traumata or from hemorrhage or infection. This condition is usually diagnosed *bursitis;* it *is* bursitis. But, for the sake of accuracy, qualifying terms such as *septic, traumatic* and *chronic villous* should be used, just as joint disturbance is not simply arthritis but is defined with terms like *septic, rheumatoid* and *osteo.*

Primary bursitis is also seen in the deeper-lying bursae, usually from trauma with hemorrhage, but is usually not diagnosed as such. Hemorrhage into a bursal sac appears to cause only a moderate amount of pain. The diagnosis of primary bursitis is usually not made in such circumstances due to concomitant trauma to adjacent structures, which figures more prominently in the diagnosis.

Subacute and chronic types of bursitis are recognized in both the superficial and deeper-lying bursae. Commonly in these types, there are thickening of the synovial wall, villous projections and cords of fibrous and synovial tissue. These types are properly called bursitis.

Cystic Degeneration

Cystic degeneration often involves a bursa. A bursa is a synovial or serous-lined, usually collapsed, cavity so situated as to allow motion between two opposing surfaces. Buck, McDonald and Ghormley[1] showed that "adventitious bursae" will form practically wherever connective tissue is subjected to excessive friction—but in this case no serosal or endothelial lining is present. In other words, it is a property of connective tissue to be able to undergo a myxomatous or mucoid degeneration. This is the kind of reaction noted in ganglia, in Baker's cysts, in cysts of the semilunar cartilages and in the ad-

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ventitious bursa of bunions, whether or not a synovial or endothelial lining is present.

The terms used in the literature on this entity are quite confusing. Baker's cysts are often called semimembranosus bursitis, for example. A number of observers have described these cystic degenerations in many and varied locations, especially around the knee and hip, as an important anatomical discovery of new bursae in the area described. These conditions would be more simply and accurately described as *cystic* or *mucoid* or *myxomatous* degeneration of whatever the anatomical structure involved. The terms *bursitis* merely confuses the issue, even though a bursa may be involved, for there is no significant amount of *itis* or inflammatory reaction. A physician who would still like to use the word bursitis somewhere in the diagnosis when a bursa of some significant size is really involved, might use the term *cystic bursitis* or *bursitis with cyst formation.*

Calcareous Deposits in Ligaments and Tendons

The word *bursitis* seems to conjure a vision of agony from an acute inflammatory reaction somewhere around the shoulder. This idea is so deeply ingrained that it appears almost impossible to overcome the habit of labeling calcareous deposits in ligaments and tendons—if they are painful and affect the shoulder—as *bursitis.* Needling or aspirating calcareous deposits is even put under the heading of Bursae in the latest fee schedules, but at least it is not confined completely to the shoulder. These calcareous deposits are still commonly spoken of as bursitis wherever they may occur, although there may not be a bursa in the near vicinity, or perhaps only a small synovial sac scarcely worthy of the name.

The clinical entity that produces the painful symptoms is the nonspecific inflammatory reaction that occurs around the calcified deposit. The pathologic change is not in the bursa, but in a ligament or tendon. No textbook or recent medical writing appears to question this fact, and yet in stating the diagnosis the term *bursitis* often is used. The bursal sac can become secondarily inflamed, but this usually happens well along in the course of the reaction, most commonly of course after direct rupture of calcified material into the bursa. But the bursa itself does not have to be inflamed in order to produce the painful symptoms. Tendons and ligaments are sensitive to pressure. They have been shown to have innervation from small nonmyelinated nerve fibers, with the usual poor localization of pain and generalized radiation. If one pinches his own biceps or Achilles' tendon, he will find that he can stand the pressure on the fold of skin lying directly over it much better than the pressure on the tendon. It is not difficult to believe, then, that the chemical furuncle with its pressure phenomena within the tendon or ligament can cause excruciating pain.

To illustrate the point, take the case of a man in his mid-sixties who, when first examined, was prostrated—almost in shock—from pain,with a reddened swollen area nine inches or more in diameter, over the medial aspect of the knee. The appearance was that of severe infection, but the patient had no fever and the leukocyte content of the blood was only slightly above the upper limit of normal range. There was a large, ropy calcification of the medial collateral ligament of the knee in the center of the inflamed area. Obviously, labeling such a condition "bursitis" or calling it by an eponym such as Pellegrini-Stieda disease, does no service to precise description.

Almost any medical practitioner who has a large office practice will see many patients with this nonspecific inflammatory reaction each year and probably will take interest in the many and various places in the human frame that it will be observed. In sightly more than eight years I have found calcified deposits with inflammatory reactions in the following locations: Rotator cuff of the shoulder, supraspinatus tendon, acromioclavicular ligament, common extensor tendon at the epicondyle, flexor carpi ulnaris tendon at the pisiform, gluteus maximus and gluteus medius tendon at the trochanter, tendons of the short external rotators of the hip at the trochanter, medial collateral ligament of the knee, the Achilles' tendon at the os calcis, plantar ligament at the os calcis. Other investigators have reported it in other locations such as the flexor profundus of the finger at the point of insertion and the flexor carpi radialis at the insertion, even in the interior longitudinal ligament of the cervical spine.

Calcification without inflammatory reactions are not concerned in the subject under discussion. They are not confused, clinically with bursitis. I have yet to see a patient walk in complaining, unprompted, that he has "a calcification."

The "Chronic Sprain"

The term *chronic sprain* is used in an attempt to cover adequately the most diverse and controversial lesion of ligaments and tendons—the lesion that needs to be described more accurately and distinguished from the entity of true bursitis. It is not a term I like or commonly use, as I will explain later, but it does cover some of the field that I wish to discuss.

The term embraces lesions in ligaments and tendons which have clinical features similar to those of reaction around calcified deposits but in which

it is impossible to demonstrate a calcified mass. A variety of names, all rather indefinite, are used to describe such lesions; *supraspinatus syndrome, scapulocostal syndrome* and *epicondylitis* are typical of the all-embracive names covering a multitude of lesions of these areas, including the entity of chronic sprain that should be set apart.

Connective tissue, wherever found in the body, has been demonstrated to undergo degenerative changes throughout a lifetime. Connective tissue subjected to stress can undergo more rapid degenerative changes, with a relationship between the amount of stress and the reparative processes, dependent upon the blood supply to the part. Connective tissues appears to be subjected to the most stress and to have the least reparative vascularity at the point of attachment to bone. Hence it is at those sites that conditions are conducive to development of an inflammatory reaction from the products of degeneration. Products of degeneration, whether they calcify or not, can be irritating to the surrounding tissue and can undergo nonspecific inflammatory reactions. Key,[2] in 1949, described fatty acids in degenerative areas which may or may not go on to calcification.

I believe that, just as microtraumata to a joint throughout a lifetime of use can produce the changes of osteoarthritis, with no pain noted unless the rate of the changes accelerates, microtraumata to connective tissue, particularly in the heavy ligaments and tendons, can bring about disruption of the fibers without pain noted until the products of degeneration accumulate faster than the reparative processes can remove them. These painful lesions, then, can be caused in some instances, I believe, by no more than ordinary stresses and strains, but may occur as the result of a single acute episode or multiple lesser insults.

Almost all practicing physicians have observed patients with complaint of a fluctuating aching pain in the shoulder, or in the elbow, or in the hip, or in the knee, with radiation often noted, the aching becoming aggravated at night, usually never acute enough to be disabling and yet annoyingly persistent for weeks or months. There may or may not be history of a traumatic episode that seems to the patient or the physician to be related. Careful palpation at the point of pain establishes the area of involvement at either the origin or the insertion of a tendon or ligament. In x-ray films, usually at least three views of the area, no calcification or other abnormality is noted. For ultimate proof of this lesion—the lesion of chronic sprain—and to confirm its position in the tendon or ligament, a small quantity of a local anesthetic can be injected (usually along with hydrocortisone nowadays) into the fibrous trigger point covering a spot no bigger than one or two centimeters. If an almost miraculous temporary relief of the entire symptom complex follows, the diagnosis is virtually certain.

The lesion of chronic sprain can calcify. Occasionally, but not usually, the reaction even without calcification is as intense as that which occurs around a calcified deposit. The flare-up of reaction following the injection and the rupturing of the products of degeneration can be intense.

The most common error that could be of serious consequence with regard to chronic sprain is to confuse it or calcified deposit in a ligament or tendon surrounding the trochanteric area of the hip, with sciatic neuritis caused by a herniated intervertebral disk and nerve-root compression. Actually the two are fairly easy to distinguish. In the case of inflammatory reaction at the trochanter commonly called gluteal bursitis, local tenderness, close to but definitely not directly over the sciatic nerve, is noted if careful palpation is done. Often there is increasing aching on bedrest. Rarely does pain radiate below the knee, the radiation commonly following directly along the iliotibial tract. Neurological deficit is not demonstrable, save for occasional atrophy of disuse. And usually the patient will maintain that the origin of pain is in the hip and will point to the greater trochanter. Nerve root compression phenomena are easily distinguishable from those of chronic sprain if one but think of the possibility.

TERMINOLOGY

As has been noted, primary bursitis, of whatever variety, can be distinguished from the entities of cystic degeneration, calcareous deposits and chronic sprains. Cystic degeneration of connective tissue likewise can be distinguished as to pathologic changes and symptoms from primary bursitis, calcareous deposits and chronic sprains. The latter two entities on the other hand are closer akin. In both, the clinical manifestation is inflammatory reaction and in both there is evidence of fibrous tissue degeneration with the products of degeneration calcifying or not, depending upon conditions which are not well understood.

I find *inflammatory tendinitis* a useful term to describe these nonspecific inflammatory reactions in ligaments or tendons. To this term, the qualifying words *with calcification* or *without calcification* are added. The term as thus constructed is serviceable enough until the cause of the inflammatory reaction is discovered. As to the redundancy of coupling *inflammatory* with *itis*, it may be pleaded that the sometimes extremely severe reaction observed in some patients may warrant this little excess of description.

Many other terms have been used throughout the years, but I find none of them quite as useful or as specific as *inflammatory tendinitis.* A term that has been long in use is *calcareous tendinitis,* and it is a good one; but what can one call the similar lesion that simply has not calcified? Although inflammatory reactions also occur in ligaments, *ligamentitis* seems too much a tongue-twister. Eponyms such as *Pellegrini-Stieda disease* are always poor as descriptive terms. And epithets like *epicondylitis* or *tennis elbow* cover more than one entity. Surely physicians can do better than simply add *itis* to the name of an anatomical part for the diagnosis and let it go at that. And surely we have enough clinical acumen that we need not resort too much to the use of the inexact *syndrome* when stating a diagnosis; if we are dealing with biceps tendinitis or a calcareous deposit in the rotator cuff, how much better to say so, precisely, than to call almost any pain located under the acromion and deltoid "a supraspinatus syndrome."

With the term *inflammatory tendinitis* used in the manner suggested, a diagnosis can be accurately stated—for example: "Inflammatory tendinitis, supraspinatus tendon, left shoulder, with calcification"; or "inflammatory tendinitis, gluteus medius tendon, right hip, without calcification." If a bursa is obviously swollen and distended secondarily, as is frequently noted in the shoulder, then the diagnosis can also include "with subacromial bursitis." This puts the term *bursitis* where it belongs, as an accompanying symptom. A diagnosis of this type needs no further explanation. It gives an accurate description of the process involved even if the process is not entirely understood. The understanding is much more likely to come if the means of communication among observers are more precise.

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No. 1 Killer

It seems that a smart automobile manufacturer, taking the long view, would sell safety to the women who control 60 per cent of all personal expenditures and influence most car-buying decisions. Witness the popularity of books on how to keep from being a widow.

Marginal Peptic Ulcer in Elderly Patients

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• **Marginal peptic ulceration occurs with sufficient frequency in elderly patients to constitute a real problem. A study was made of 22 patients more than 65 years of age in whom such ulcers developed.**

Marginal ulcers in elderly patients are frequently associated with dangerous complications such as massive hemorrhage or perforation.

Advancing age does not necessarily reduce the tendency to marginal ulcer formation.

Such operative procedures as gastroenterostomy without vagotomy, and minimal gastric resection, do not afford adequate protection from marginal ulceration in elderly patients.

When marginal ulcers occur in elderly patients, adequate gastric resection (70 to 75 per cent) or vagotomy in combination with resection is usually necessary.

MARGINAL PEPTIC ULCERATION may follow gastric operation of any type and may appear in any age group. It tends to occur near gastrointestinal stomas, the most common site being on the jejunal side of a gastrojejunostomy. Marginal ulceration occurs most often in patients with duodenal ulcer who have been treated by gastrojejunostomy alone. The ulcer recurrence rate in such persons may be as high as 34 per cent in long-term follow-up studies.[9,12] Marginal ulcers also occur following gastric resection, but the incidence varies from 1 per cent to 5 per cent.[11,15] When marginal ulceration occurs after gastric resection it generally appears within five years of the operation, whereas if it occurs after gastrojejunostomy it may not develop until ten years or more after operation.[15] Although marginal ulcers are rare following resection for gastric ulcer they do occasionally occur. In recent years vagotomy combined with gastrojejunostomy or pyloroplasty has been widely employed for the treatment of duodenal ulcer and the marginal ulcer rate following this procedure appears to be approximately 8 per cent based on five to ten year follow-up periods.[7]

Marginal ulcers are frequently associated with severe complications which are frequently resistant to conservative management. Pain and bleeding are the outstanding clinical manifestations of recurrent ulceration. Bleeding, when it occurs, is frequently massive. Other serious complications such as perforation and gastrojejunocolic fistulas may develop.

During recent years increased emphasis has been placed on the surgical problems of elderly patients, and important among these is the problem of gastroduodenal and also marginal peptic ulceration. More and more frequently elderly patients are being seen with complicated peptic ulcer problems requiring surgical treatment. The marginal ulcer problem in the older population has not been given sufficient consideration due to the fact that peptic ulceration, in general, has been assumed to be a disease largely confined to younger and middle-aged persons. The purpose of this paper is to present an analysis of a series of patients in the older age groups with marginal ulcer problems.

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From the College of Medical Evangelists School of Medicine, and from the Surgical Services of the White Memorial Hospital and the Los Angeles County General Hospital.

ANALYSIS OF STATISTICAL MATERIAL

A study was made of 22 patients 65 years of age or over who were treated for marginal ulcers at the White Memorial and Los Angeles County General Hospitals from 1947 to 1957. In 19 cases the ulcers were gastrojejunal, in three gastroduodenal. The gastroduodenal ulcers followed pyloroplasty and vagotomy in two patients and Billroth I gastric resection in one patient. The three cases of marginal ulcer following gastroduodenal anastomosis were not included in the analysis.

Age

In the group of 19 patients with gastrojejunal ulcer the age range was from 65 to 80 years. There were six cases in the age group of 65 to 69 years and 13 cases in the age group of 70 years or over. According to Kiefer and McKell,[8] the first symptoms of peptic ulceration may appear in patients who are 70 years of age or more. In the present series there was one case in which initial ulcer symptoms began at the age of 72 years, and in six cases the operation preceding the gastrojejunal ulcer was performed in patients who were then over 65 years of age. Davey and O'Donnell[4] could find no evidence that older people are less liable to marginal ulceration. In a recent review of peptic ulcer disease in elderly patients at the Los Angeles County General Hospital,[14] it was found that there was a high incidence of lethal complications, and that advancing age afforded no safeguard against their occurrence.

Time Interval

It has been stated that the interval between partial gastrectomy and the development of marginal ulcer is shorter than that after gastroenterostomy. In the present series 16 patients had a marginal ulcer occurring after gastroenterostomy and three patients had ulceration following the Billroth II partial gastrectomy. There was no difference in the time interval following the two types of operation, but the group of cases is too small for statistical significance. The interval from operation to the appearance of marginal ulcer varied from one month to 32 years. In 11 cases the interval was less than 20 years, and in eight cases it was 20 years or more.

Symptoms and Complications

The complications of marginal ulcer are similar to those of peptic ulcer in general, except that the incidence of hemorrhage is greater. In nine cases in the present series the complaint that brought the patients to the hospital for treatment was hemorrhage, and in four of these cases it was severe enough to be classified as massive. Severe pain does not appear to be a common complaint in elderly patients with marginal ulcer except with perforation, which occurred in four cases. In one patient there was stomal obstruction due to an ulcer in the proximal loop of the jejunal anastomosis. It was severe enough to require surgical correction. One patient had a gastrojejunocolic fistula which occurred 17 years after gastrojejunostomy. The symptoms in this case were bloody diarrhea and pronounced loss of weight.

Diagnosis

The presence of marginal ulcer is readily suspected when a patient with a history of operation for peptic ulcer complains of abdominal pain or melena. To confirm the diagnosis is more difficult, for physical symptoms may be minimal and x-ray examination often is inconclusive. In more than half of the cases in the present series the results of gastrointestinal x-ray studies were negative.

Pathologic Features

The marginal ulcers were usually either at or in close proximity to the stoma on the jejunal side. In all cases the ulceration was within 2 cm. of the stoma. In a case reported by Thompson,[15] however, the ulcer was located in the jejunum 15 cm. from the stoma. Although ulcers in the jejunum usually occur in the efferent loop, the lesion was in the afferent loop in one case in the series. In four patients the ulcer was on the gastric side of the stoma. The largest ulcer in the series, about 6 cm. across, was in this location. Most of the ulcers were 1 to 2 cm. in diameter; the smallest was 0.5 cm. Most of the ulcers were single, and most were deep and penetrating, but a few were only superficial mucosal erosions.

Usually at operation an area of induration was noted at the stomal site and in some cases a definite ulcer crater could be palpated. In other cases, however, it was necessary to open into the stomal area to determine the presence of ulceration.

Treatment

Of the 19 patients treated for gastrojejunal ulcer, three were not operated upon. Two of them had mild pain which responded to conservative therapy; and the other, an 80-year-old woman with perforation (proven later at autopsy) entered the hospital in terminal condition and died within 24 hours. The rest of the patients received surgical treatment and four types of operative procedures were used.

Vagotomy. Vagotomy alone was performed on four patients. One patient had a mild recurrence of bleeding after an abdominal vagotomy for stomal ulcer following gastroenterostomy. Another patient died from persistent gastrointestinal hemorrhage due to a stomal ulcer occurring after pancreaticoduodenectomy; the intestinal reconstruction after this procedure was unfortunately arranged in such a way as to divert the alkaline juices from the gastrojejunal stoma. The hemorrhage was not relieved after thoracic vagotomy. The other two patients had satisfactory results.

Simple closure of perforation. This procedure was performed on three patients with acute perforation and in one instance it was combined with vagotomy and in another it was followed in two years by vagotomy.

Pyloroplasty and vagotomy. It was possible in three patients to resect the gastroenterostomy stoma with the ulcer, suture the gastric defect and reestablish continuity of the jejunum. Vagotomy with pyloroplasty was then added to control the basic ulcer disease. Satisfactory results were obtained in each of the cases.

Partial gastrectomy. There was no evidence of recurrence in six cases after subtotal gastrectomy (60 to 80 per cent of the stomach was removed). There was a recurrence after nine years in a patient who had been treated by hemigastrectomy and vagotomy. From observations made on this small series of patients it would appear that adequate gastric resection (70 per cent) could be expected to give the best results in treating stomal ulcers. However, many of the patients in the older age group are such poor surgical subjects that subtotal gastrectomy cannot always be performed without

unwarranted risk. Consequently such procedures as vagotomy and pyloroplasty must sometimes be used even if there is a greater danger of recurrence present.

Mortality

The lethal complications in this series were perforation and massive hemorrhage, which caused three deaths in 19 cases. This mortality rate was about the same as that previously reported in complications of peptic ulcer in elderly patients.[14]

The dangers of hemorrhage and hemorrhagic shock are well recognized. Since perforation in elderly patients may not produce the severe pain and pronounced acute abdominal symptoms that it does in younger patients, treatment frequently is delayed.

REPORTS OF CASES

CASE 1. A 71-year-old white man was admitted to the hospital on December 12, 1953, with complaint of epigastric pain, hematemesis, tarry stools and weakness. Two years before, the patient had a perforated duodenal ulcer without previous ulcer history. The perforation was treated by simple closure and the postoperative course was satisfactory. About four months before the present admittance, a posterior gastroenterostomy was done because of pyloric obstruction.

Upon examination, poorly localized abdominal tenderness was noted. The blood pressure was 110/70 mm. of mercury, the pulse rate 100. The hemoglobin content of the blood was 7 gm. per 100 cc. A diagnosis of bleeding gastrojejunal ulcer was made and operation was performed on December 23, 1953. A 1.5 cm. ulcer was found in the distal loop of the jejunum 1 cm. from the gastroenterostomy stoma. Gastrotomy was performed to identify the lesion and a vagotomy done for definitive treatment. The patient made an excellent postoperative recovery.

Comment. In this case the first symptoms of duodenal ulcer occurred after the age of 65 years. Also to be noted is that gastroenterostomy without vagotomy at this age as well as in younger patients is not adequate to prevent the occurrence of marginal ulcer.

* * *

CASE 2. An 80-year-old white woman complained of epigastric pain for five hours before entry into the hospital on March 4, 1957. She gave a history of previous attacks of abdominal pain for 14 years. In 1948 cholecystectomy was performed and also an anterior gastroenterostomy because of duodenal scarring.

Upon examination tenderness in the mid-abdomen and hypoactive peristalsis were noted. A previous gastrointestinal x-ray taken in 1950 showed a deformed duodenal bulb and a partially obstructed gastroenterostomy, but no ulcer crater was seen at the stoma.

Shortly after entry into the hospital the patient went into shock and did not respond to supportive measures. She died within 24 hours. At autopsy a 2 cm. perforated jejunal ulcer was found just distal to the gastroenterostomy stoma.

Comment. It is possible to have perforation of a duodenal or gastrojejunal ulcer in any age group. Although uncommon, such perforations may occur in elderly women as well as men. The preoperative diagnosis was difficult to make in this case because of the lack of characteristic physical signs, which is frequently the case in elderly patients. The diagnosis of perforated peptic ulcer in the older age group is often missed, which is a factor in the high mortality rate.

* * *

CASE 3. A 67-year-old white man entered the hospital on May 18, 1955, with complaint of bloody diarrhea for two months and of a 16-pound decrease in weight in one year. He gave a history of perforated peptic ulcer in 1928 and of gastroenterostomy in 1938, but the patient did not know the reason for the operation.

No abnormalities were noted upon physical examination. A roentgenographic study showed a gastrocolic fistula near the gastroenterostomy stoma.

At operation the gastrojejunostomy stoma was resected, the fistula was closed and vagotomy with pyloroplasty was performed for definitive treatment. The operative procedure was well tolerated, and the patient made a satisfactory recovery.

Comment. Operation for gastrojejunocolic fistula in elderly patients entails a high mortality risk, and staged operations are often necessary. The use of vagotomy with pyloroplasty, an operation of lesser magnitude than gastric resection, gave good short-term results in this case, but a longer follow-up period is necessary to evaluate the procedure.

* * *

CASE 4. A 71-year-old white man entered the hospital on December 4, 1956, with a history of peptic ulcer treated 14 years previously by gastroenterostomy. Nine years previously he had had abdominal pain, vomiting and melena, and x-ray examination at that time showed an ulcer crater 1 cm. in diameter at the gastroenterostomy stoma. The patient was successfully treated by hemigastrectomy and vagotomy.

The present hospital entry was for small bowel obstruction, which at operation was found to be caused by old postoperative adhesions. Upon examination of the previously made gastrojejunal anastomosis, a 2 cm. ulcer crater was observed on the

posterior wall of the jejunum adjacent to the stoma. No surgical treatment was attempted at the time because of the poor condition of the patient. Conservative therapy, however, gave the patient complete relief.

Comment. This recurrence of peptic ulceration at the age of 71 years even after hemigastrectomy and vagotomy again emphasizes the persistent nature of peptic ulcer disease in many elderly patients.

DISCUSSION

Interest in the study of elderly patients with marginal ulcer was stimulated by the frequent recommendation in medical literature and current textbooks on surgery, of the use of gastroenterostomy alone in the treatment of aging patients with duodenal ulcers.[1,2,5,6,10] It has been thought to be a relatively safe and effective procedure in this age group, particularly when used for stenosing, obstructing lesions. Our hospital records did not show any patients over 65 years of age with marginal ulcers until the past ten years. The present series of cases accumulated during the past ten years would tend to suggest that marginal ulceration is occurring or is being noted more frequently than before and that increasing age does not offer any protection against the occurrence of a marginal ulcer or any of its complications. It is not generally recognized that the occurrence of peptic ulcer, in general, in patients over 60 years of age is nearly the same as it is in younger age groups. Although it has been shown that there is some decrease in gastric acidity in older patients, one might well postulate that decreased local tissue resistance to peptic ulceration might be present in the older aged groups. It should be emphasized that six patients in this series had gastroenterostomy alone after the age of 65 years and that marginal ulcers subsequently developed.

The severe and often lethal complications of marginal ulceration, such as massive hemorrhage or perforation, present special problems in diagnosis and treatment in older patients. Such complications are not well tolerated by them and a high mortality rate may result. Likewise emergency surgical operation is poorly tolerated by older people, and many times inadequate operative procedures are done presumably because of the poor-risk factor. It is known, however, that major operative procedures such as gastric resection are well tolerated by aged patients if they are done under elective circumstances.[4]

The prevalent belief that marginal ulcers are rare in older patients has encouraged the continued use of this inadequate operation. It would appear that peptic ulceration in elderly patients should receive prompt surgical attention if conservative management is not quickly successful. Major operative procedures such as subtotal gastric resection (70 per cent) and vagotomy with pyloroplasty or gastrojejunostomy may be performed on older patients with a low mortality rate if they are done on an elective basis.

Peptic ulceration which occurs near an anastomotic line between the stomach and the small intestine, whether it be duodenum or jejunum, may also be referred to as "marginal."[15] The group of three patients in this series with marginal ulceration (occurring after vagotomy and pyloroplasty in two cases and after Billroth I type gastric resection in one case) is of course too small for any credible evaluation. However, inasmuch as these two procedures have been commonly employed during recent years in the treatment of duodenal ulcers, one would anticipate that an increasing number of marginal ulcers of this type will be seen. It is probable that the recurrent ulceration after vagotomy and pyloroplasty was the result of inadequate vagotomy, although this was not actually demonstrated in these two patients. It appears that the marginal ulcer rate following Billroth I end-to-end type gastric resection for duodenal ulcer may be excessive. The treatment of a marginal ulcer, once it has occurred, in an elderly patient is essentially the same as it would be for a patient in any other age group. When a marginal ulcer occurs after gastroenterostomy alone, the procedure of choice is resection of the anastomotic area, followed by adequate (70 to 75 per cent) gastric resection. If the marginal ulcer appears following gastric resection, vagotomy is often sufficient. If the resection was inadequate in amount, however, a re-resection may also be necessary. In certain especially poor-risk patients it may be necessary to use procedures of lesser magnitude than subtotal gastrectomy, recognizing that the chance of recurrence is present.

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Employment of Persons with Epilepsy and Heart Disease

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• **Among persons who are often considered handicapped so far as employability is concerned are two groups deserving of special attention—persons with heart disease because of the ever-increasing number of them, and persons with epilepsy because of the extreme difficulty they have had in getting suitable jobs.**

A considerable number of persons with cardiac disease and a lesser number with epilepsy have been employed by the California Division of Lockheed Aircraft Corporation during the past ten years. It has been the experience there that the employment of such persons, once they are placed in jobs compatible with their physical limitations, has not resulted in unduly increasing the company's liability or labor cost and has not created operational problems of major proportions or jeopardized anyone's safety.

Considerable research and effort are being expended by governmental and other agencies and associations to further enhance the safe placement of such persons. It is believed that these efforts, if coupled with realistic interpretation of the Workmen's Compensation Act in this regard, will bring about more liberal and widespread acceptance of persons with heart disease and epilepsy by industry.

GREAT EFFORTS have been made by many persons and agencies during recent years to alert industry to the employment potentials of physically handicapped persons. Among the so-called handicapped persons are two groups that deserve special attention, the patients with heart disease because of their ever-increasing numbers and epileptic persons because of the extreme difficulty they have had in obtaining employment in the past.

Lockheed Aircraft Corporation has had considerable experience in the employment of both groups and since the medical department is intimate with the problems, we welcome this opportunity to discuss some of our experiences and observations and to describe our procedures and philosophy relevant to the medical aspects of placement of physically substandard employees.

We have been most fortunate in our association with a company noted for its humanitarian and progressive employment policy. This policy, as applied to persons with heart disease, was stated thus by one of our corporate officers in a publication entitled *These Hands Are Able:* "Industry today is hiring more and more handicapped workers. Many are cardiacs. Employers who know the facts about persons with heart disease, who apply proper placement, are making intelligent use of this available work force. At our company, cardiacs have proved capable and efficient. Placed in a job within their capacity, they create no undue liability or safety risk."

This philosophy is incorporated in company directives, which state in substance that it shall be the company's policy to employ physically handicapped persons and permit employees recovering from disabilities to return to work when to do so does not (1) jeopardize either their own safety and well-being or that of their co-workers; (2) unduly increase the company's liability or labor costs; or (3) create operational problems of major proportions.

These principles were translated into workable realities under the capable and sympathetic directorship of the late Dr. Fenn E. Poole. How well he accomplished his job is evidenced in the company's medical rejection and cancellation rate, which is among the lowest in industry. Less than 2 per cent of all applicants technically qualified for available positions are disqualified because of medical evaluation.

Our medical evaluation and placement system operates on the basic philosophy of maximum utilization and minimum wastage of manpower. We feel that even the limited or physically substandard worker, regardless of his handicap, once properly matched to a job ceases to be handicapped for that job and can perform as efficiently and safely as the average worker. This program of selective placement has not, during our association with Lockheed, resulted in increased compensation costs, increased risk to the company or an increased accident rate.

The preemployment or, more descriptively, placement examination is the means most commonly used to evaluate the physical capabilities and/or limitations of the employee and also serves as a baseline and detailed record of his health inventory at the

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time of hire. The medical history is, or should be, the most important phase of the entire examination procedure. At least 75 per cent of all existing disease can be suspected on the basis of symptomatology or information volunteered by the applicant.

Refinements in the placement examination should be directed primarily to eliciting a more accurate and detailed history. This does not necessarily imply the addition of dozens of complex questions disguised to trap the applicant into an admission. Rather it indicates the necessity of educating him in the realization that such information is needed to protect his health and ensure his safe placement.

We use an examination form containing fifty to sixty questions with additional space for recording data from a series of four physical examinations and the physicians' remarks. The examination form serves as a jacket for x-ray films, laboratory reports and pertinent medical correspondence.

A routine examination includes examination of the applicant's vision, using an American Optical Company Sightscreener, urinalysis and stero 4 by 5 inch x-ray films of the chest. Audiograms are made on all employees and additional laboratory studies are ordered in the cases of applicants for certain kinds of jobs—such as painting and de-greasing. Other studies such as electrocardiograms, skeletal roentgenography and blood chemistry determinations, are performed only when indicated on the basis of physical findings or significant history.

CLASSIFICATION OF LIMITATION

The results of the examination and the physician's recommendations must be transmitted in a simple and easily interpretable form to the employment interviewer and eventually to the employee's supervision to make sure any restrictions are complied with. For this purpose a classification system, using six major categories, was developed. It expresses the physical activity, condition or environment to which the employee must not be exposed, rather than classifying him on the basis of positive qualifications for a single job. Since about 75 per cent of our handicapped personnel require limitation with regard to only a single function, this method has proved very effective. We find our system affords flexibility in intradepartmental utilization of the worker and facilitates interdepartmental transfer.

CLASS I. *No hazardous machinery.* Employees having serious visual defects, diabetes, epilepsy, high blood pressure or heart disease may be placed in this category.

An employee limited to Class I is not permitted to operate hazardous power-driven machinery at any time. *Hazardous* power-driven machines include: Saws, routers, lathes, shapers, mills, grinders, drill presses, jig borers, drop hammers, punch presses, shears, radial drills, and others of the kind.

Employees limited to Class I are forbidden to use any of the above machines. In addition they are not permitted to operate a commercial vehicle such as a highway truck, or an in-plant truck such as a goose, tug or tractor, nor an overhead crane if cab-controlled. *Nonhazardous* machines which those limited to Class I may operate, if proficient, include rivet guns, nut runners, hand-drill motors, burring tools and others.

CLASS II. *No heavy lifting.* Employees with heart disease, back or joint trouble, improperly supported hernias or high blood pressure may be placed in Class II.

Men limited to Class II are not permitted to lift more than 25 pounds at any time. Women limited to Class II are forbidden to lift more than 10 pounds and should not be required to do constant lifting.

(Women employees without medical limitations are forbidden to lift more than 25 pounds under any condition. No woman employee shall be requested or permitted to carry any object weighing 10 pounds or more up or down any stairway or series of stairways that rise more than 5 feet from its base.)

Pushing or pulling weights requiring draw bar force in excess of 50 pounds is prohibited for male employees with Class II limitation. Female employees with Class II limitation are restricted to draw bar force of 25 pounds.

CLASS III. *Ground level work.* Employees with high blood pressure, diabetes, epilepsy and heart trouble, who are susceptible to bouts of dizziness or fainting spells may be included in this class. In addition, those having a deformity or disease of the spine or lower extremities may require this limitation.

An employee limited to Class III is not permitted to work on ladders, scaffolding, airplane wings or temporary jigs or in any other unprotected elevation.

Second and third floors of permanent, protected jigs and buildings, and the lower three steps of a five-step ladder are not considered hazardous for Class III limitation. Walking up and down stairs to and from such places of work is not a violation of the limitation.

Movable steps, ramps or raised platforms designed to facilitate working in permanent jigs and fixtures, such as those found on the assembly lines are not considered hazardous for Class III limitation.

CLASS IV. *Avoid contact with or exposure to specified agent.* Employees suffering from chronic sinus trouble, bronchitis, asthma and skin allergies may require this limitation.

An employee limited under Class IV must be placed where he will not come into contact with the specific agent or agents designated by the medical department. Irritants are generally chemicals, fumes, dusts or other agents that may be inhaled or come into direct contact with various portions of the body.

Allergic responses to these irritants vary between persons and sometimes occur without prolonged exposure or contact. The presence of the irritant in the air may be sufficient to produce a reaction.

CLASS V. *No extensive walking or standing.* Employees suffering from back disorders, lower extremity defects and varicose veins may require this limitation.

An employee limited to Class V is not permitted to walk or stand more than 50 per cent of the working shift. Sedentary bench or desk work is best for employees limited to Class V. Work permitting alternate sitting and standing is permissible and frequently desirable.

CLASS VI. *Miscellaneous.* Limitation to Class VI indicates the presence of a physical condition which must be shown special consideration. Employees with ear disease, nervous disorders, chronic back distress, bursitis and allied conditions may require this limitation.

All conditions which require limitation and are not included in the first five categories are placed in this class. When this class is used, the specific nature of the limitation is stated.

For example: An employee who should not be exposed to noise would be restricted to Class VI, "Not to work in an area where noise levels are above 95 decibels"—drop-hammer operator, riveter, aircraft mechanic. Or a woman with a severe strain of the elbow might be limited as follows: Class VI, "Not permitted to use hand tools requiring squeezing."

All medical information concerning the employee is considered privileged and confidential, and we forward only the numerical classification of limitations to the employee's department. To facilitate compliance and enforcement of the limitations, it was decided to display the limit by number on the employee's badge. Contrary to the fears and expectations of some, open identification of our physically substandard employees did not arouse a storm of complaints; a few employees asked that it be removed, but it has been generally accepted. The on-the-job supervisors of handicapped workers have found it fairly simple to comply with limitations when they are constantly in evidence, and safety engineers are able with a minimum effort to ascertain that a worker is not placed in a job proscribed for him.

Job evaluation is the second major component in the process of selective placement, as it defines the physical requirements for each job as well as the potential risk or hazard involved. From the medical standpoint, an ideal job evaluation is one that defines exact energy requirements as well as the type and degree of body movements and environmental exposure. However, since few routine physical examinations are designed to measure energy potentials, less refined procedures are and can be utilized. The job analysts in our wage and salary section outline the basic and overall technical and physical requirements of each job. The safety engineers, in their own studies, determine the physical risks and potential hazards associated with each operation. Finally, in making requisitions for workers, each department head must complete a detailed form in which he lists the degree and type of physical activity for the specific job and the environmental conditions to be encountered. This information is conveyed to the employment or placement interviewer, who in turn receives the results of the medical evaluation from the physician. It is, of course, the sole and inviolate responsibility of the medical department to determine the extent of body function or physical stress that is safe for the applicant. The placement expert can then extrapolate the medical evaluation in terms of fitness for any job.

We have made several recent studies of our employees who have heart disease or epilepsy. Out of a total work force of 31,000 we were able to identify 725—or 2.4 per cent—employees with cardiovascular disease. This indicates the number of known cases brought to the attention of the medical department. Of the 725, 301 had hypertension, 220 had some form of coronary artery disease, 40 had rheumatic heart disease, 13 had valvular cardiac disorders, 57 had heart disease of undetermined origin and the remainder other less prevalent forms of vascular disease.

WORKERS WITH HEART DISEASE

Dealing with workers with heart disease raises some interesting problems in employment. Heart disease is fairly prevalent among the working population and is the cause of 45 per cent of all adult deaths, the majority attributable to coronary artery disease. Other cardiac conditions, such as congenital defects, valvular disorders, rhythmic disturbances and hypertensive heart disease frequently cause abnormalities that can be detected during a cursory examination of the heart and blood pressure, but coronary artery disease is insidious in that pathological changes frequently occur before any abnormality is demonstrable.

The incidence of secondary occlusions in persons

with coronary artery disease is high, and a damaged heart muscle implies a limited functional reserve. Lightheadedness and faintness may occur and under conditions of extreme physical exertion or emotional stress coronary artery spasm may be precipitated in diseased blood vessels. For these reasons an accurate diagnosis and physical evaluation is essential so that the applicant or employee may be placed in a job involving minimal risk to himself and protecting the employer against unjust and extensive liability.

At Lockheed, once cardiac disease has been diagnosed, work limitations are imposed in accordance with our placement standards. Usually, lifting weights of more than 25 pounds is forbidden, and the employee may work only at ground level. In more severe cases it is necessary to impose limitations ensuring minimal standing, no operation of hazardous machinery and occasionally no work entailing unusual mental stress.

All employees with limitations for heart disease are reevaluated six months after employment to ensure adequacy of placement. The frequency of additional examinations is determined on an individual basis. It would, of course, be ideal to precisely determine the energy potentials of each applicant with heart disease and to match this with individual job requirements. In a company such as Lockheed, with many hundreds of job classifications and numerous applicants with heart disease, it would be difficult to preform these precise measurements. Placement philosophy, therefore, entails conservative limitations for all workers with heart disease to ensure employment well within their reserve potentials.

It is occasionally necessary to reject applicants with severe cardiac conditions as being medically unsuited for work of any kind. Applicants who have had multiple coronary occlusions or who have active heart failure and persons who have severe congenital defects and are in imminent danger of heart failure are deemed unemployable. Employees in whom heart disease develops after they are hired are required to obtain maximum treatment before they are permitted to return to work. Careful cardiac evaluation is then instituted and every attempt made to return the employee to his former position or, if that is not compatible with his work restrictions, to retain him in an equivalent capacity.

It has been our experience that once a person with heart disease is placed in a suitable job, he ceases to be handicapped. The work efficiency and output, the accident rate, attendance and motivation of such persons are at least as good as for other employees. They pose essentially the same problems as any physically substandard employee with regard to loss of flexibility for job transfer.

Approximately 18 per cent of the employees at Lockheed are physically limited. Almost 20 per cent of those who are limited have cardiovascular disease. Many of them had the disease at the time of employment. This raises a frequently asked question: Is the employment of such persons of major importance in increasing sickness and life insurance rates, and what is their liability potential relative to workmen's compensation?

Of approxmately 40,000 men and women hired at Lockheed during the years 1951 and 1952, 19 died of coronary artery disease and two of other cardiac conditions. In only four cases was a history of heart disease elicited at the time of hiring, and in only three of these were objective physical findings present. Heart disease accounted for 48 per cent of deaths in the entire personnel, a figure which compared closely with mortality data of the general adult population in Los Angeles County. In view of this close approximation, we concluded that working stress at Lockheed had no special influence upon the death rate.

Ninety-five per cent of all employees who died of heart disease were more than 40 years of age and the median age was 53. The great majority of deaths occurred during nonworking hours. In no case was a serious question raised concerning compensability under the Workmen's Compensation Act. It has been our policy to have a visiting nurse talk with the immediate relatives as soon as possible after the death of an employee and to act as the company representative in all matters pertaining to the death, including those pertaining to pecuniary benefits. This practice tends to establish a good rapport with the family and is invaluable from a public relations standpoint. It tends to negate subsequent misinformation conveyed to relatives from many poorly informed sources.

MORE SENSITIVE EXAMINATION NEEDED

Since fewer than 20 per cent of employees who died of heart disease within two years after they began work were known by the company to have cardiac abnormality at the time they were hired, it is obvious that refinement of the examination procedures is needed. Routine electrocardiograms on all applicants are, we feel, not justified as a mass screening procedure. However, for applicants over 40 this test may be economically and medically feasible. A one-lead electrocardiogram, using the first lead recorded by a direct writing machine, has been used in some clinics as a rapid screening procedure, although it is of doubtful value since some conditions (posterior coronary disease) are not manifested in Lead 1.

Within the last several years additional tests have

been advocated for diagnostic and prognostic purposes. Of these, the Gofman test or atherogenic index determination, the blood cholesterol determination and the microchylom index or dark field test of Griffith have received considerable attention. We have explored the possibility of using these procedures but find them to be impracticable for routine screening purposes. Several of the tests are expensive, complex and time-consuming. It is doubtful that they contribute more, prognostically, than does a history of familial heart disease. A quick-acting, economical and simple screening test for coronary heart disease remains to be developed.

Considerable research has been carried out in an attempt to precisely evaluate persons with heart disease for employment. The United States Department of Public Health recently sponsored an extensive cardiac rehabilitation project which was conducted, in part, at Lockheed. One objective of the study was to accurately determine the energy requirements of different jobs and define the oxygen consumption per unit of body area for normal persons and persons with heart disease. This phase of the program was conducted entirely on Lockheed worker volunteers. The second phase consisted of a series of tests, including pulmonary compliance studies, determinations of chemical contents of the blood, treadmill tolerances, electrocardiograms and others, performed at a special laboratory in one of the largest hospitals in Los Angeles. On the basis of these studies, it was hoped to develop a physical fitness index by which a subject could be classified on the basis of his ability to perform muscular work. It was hoped that the physical fitness indices might be standardized within a normal range and that an abnormal index for persons with heart disease could be extrapolated in terms of the known energy requirements for various jobs.

Of more immediate importance has been the establishment of the Work Classification Unit of Cardiacs in Industry Committee of the Los Angeles County Heart Association. The staff consists of several cardiologists, a job analyst, a social worker and an administrator. Persons with heart disease in need of evaluation are referred to the Work Classification Unit, where complete studies are performed. These include medical and emotional evaluation, social history and job analysis. Upon completion of the studies, a conference is held between the physicians and their co-workers, and a decision is rendered as to the subject's ability to perform a given job. Through this means, a just, honest and scientific diagnosis is reached and realistic recommendations made concerning the work capabilities of persons with heart disease. At present the unit accepts only persons who meet the following requirements: They must wish to have an evaluation of their work potential, must be referred by an industrial physician, must have had a diagnosis of heart disease, must be employed and have an employment problem with regard to the cardiac abnormality. This service is offered without charge to the subject or the employer. It is an initial effort and, unfortunately, must be limited in scope of selection, although it is hoped to extend the program later to provide a greater degree of coverage.

COMPENSATION FACTORS

On the darker and more pessimistic side are recent interpretations of the Workmen's Compensation Act by the Industrial Accident Commission and the highest courts of California which threaten to retard the progressive employment practices of many companies—decisions which imply, in effect, that almost any disease or injury occurring while the employee is on company premises is compensable. It now appears that an injury received in a fall to the floor, regardless of the cause, may be compensable. A person with heart disease who loses his balance as a result of lightheadedness or vertigo, or one who falls as a result of a coronary seizure, can now incur a potentially compensable injury.

Heart disease is a progressive disorder entailing the possibility of unpredictable calamity—which must be recognized in any just compensation law. Much confusion exists concerning definition of such words as *disease* and *disability*, and clarification is needed. Compensation adjudication procedures must be corrected to fairly limit the liability now assumed almost entirely by the employer. Secondary injury laws have proved ineffective in practice and the Subsequent Injuries Fund in California, which is useful in some disorders, is of doubtful value in cases of heart disease. It has been aptly stated that current decisions by the courts and the Industrial Accident Commission have done more to hurt the cause of persons with heart disease, epilepsy and other chronic diseases than all the good accomplished by the arduous efforts of many fine organizations, companies and individuals. What is most distressing is the effect upon those far-sighted companies with the most liberal and progressive policies who are most vulnerable by virtue of continuing these policies in force.

In 1953 a special attempt was made at Lockheed to gather data on all known epileptics employed in the company and subsequently to evaluate their progress. One of the objectives of the study was to determine whether it was possible to remove all work limits after a number of years free of seizures. During a three-year period data was kept on 63 epileptic employees, 15 of whom were military veterans. The

average age was 37 years. Only one-third of these employees admitted they knew at the time they were hired that they had epilepsy. In addition, five applicants who identified themselves as epileptics at the time of applying for jobs could not be employed because of medical limitations. Among the 63 who were employed, 14 had been with Lockheed more than ten years. The average duration of employment was 5.2 years. Seven of the 63 were females. The majority of subjects had grand mal epilepsy. Twenty-eight were under the regular care of a physician and taking medication. Thirty-six were employed in medium or upper income positions.

Twenty-two left the employment of the company during the period of the study. Seven voluntarily terminated employment, four were laid off during production cut-backs, and four were discharged by the company, two of them because seizures were not adequately controlled by medication. The remaining seven either transferred to other divisions or did not return from sick leave.

Applicants for employment are encouraged to reveal that they have epilepsy. We never arbitrarily reject epileptic persons for employment but attempt to place them in positions compatible with their physical limitations. This generally involves the application of limitations prohibiting these persons from operating power-driven machinery and/or driving vehicles and from working on unprotected overhead platforms. This, we feel, tends to minimize the possibility of serious injury in the event a seizure occurs without sufficient forewarning to permit the subject to sit down. Seizures of the grand mal or Jacksonian type, when they occur without foreknowledge, tend to have an adverse effect upon the morale of fellow workers and to cause work delays.

It is extremely difficult to diagnose epilepsy on the basis of the preplacement physical examination. In the majority of cases the medical department first becomes aware of the existence of epilepsy as the result of a spontaneous seizure occurring during employment or by information obtained from other sources. Occasionally a first attack of epilepsy will occur in a young adult after his employment, but in the majority of cases the employee deliberately falsifies his medical history.

Falsification of medical records at the time of original employment is cause for dismissal. Occasionally this prerogative has to be exercised by the company when applicants deliberately refuse to allow the medical department to evaluate their disorder and properly place them in a suitable job assignment.

In our experience, for the most part, epileptic employees under adequate medical control perform as effectively as the majority of other employees. We have found no unusual incidence of mental deterioration or reduced cerebral capacity. To the contrary, several important positions are being capably filled by epileptic persons. We have noted a tendency for the frequency of seizures to decrease with time in almost all types of epilepsy, with the possible exception of Jacksonian seizures. The latter are a most difficult problem, in that they are hard to control and are frequently precipitated by relatively minor trauma to the head.

There are a number of groups in the Southern California area who are active in the rehabilitation of epileptic persons. One such group, Epi-Hab, sponsors a workshop in Culver City and trains epileptic persons as skilled machine operators and technicians. Medical surveillance is provided at all times. Although seizures among the workers are not uncommon, there have been no injuries owing to the seizures despite the fact the subjects are operating potentially hazardous power-driven machinery.

Among the epileptic persons at Lockheed are engineers, fabricators, stock clerks and shipping clerks, electricians, assemblers, inspectors, painters, mechanics, stenographers, executives and maintenance men.

As with other employees who have medical limitations, the chief operational problem with regard to workers who have epilepsy is loss of flexibility in job transfer. Perhaps more important in employment consideration is the possibility of an employer's incurring excessive potential liability as the result of injuries that occur during seizures. As in the case of employees with heart disease, it appears that injuries caused by the worker's falling, regardless of the idiopathic nature of the seizure, are compensable under the Workmen's Compensation Act. Hence employers are extremely reluctant to hire epileptic persons. The solution once again lies in a more realistic understanding of the problem, especially as it pertains to the legal responsibility of the employer. The majority of epileptic persons should have no difficulty in getting jobs for which they have the necessary training and technical skill, provided their seizures are under adequate control and hiring them involves the employer in no major medical/legal problems.

2555 North Hollywood Way, Burbank (Lorbeer).

Acute Barbiturate Intoxication

A Case with Extremely High Content in the Blood and Protracted Coma with Recovery

RICHARD I. CRONE, Colonel (MC),
DAVID E. JOHNSON, Captain (MC), and
ERIC I. ANDERSON, Captain (MC), U. S. Army

THE WIDESPREAD USE of the barbituric acid derivatives has given rise to the very serious problem of acute barbiturate poisoning resulting from both accidental and deliberate overdosage. Robie[15] said that the suicidal deaths from barbiturate intoxication have reached astounding proportions, with a 375 per cent increase in New York City, a 670 per cent increase in Los Angeles, and a 1,300 per cent increase in Chicago over the past seven, three and eight years, respectively. We wish to report a case of severe prolonged coma with complications successfully treated with supportive and analeptic therapy. In this case the plasma phenobarbital level was 21.6 mg. per 100 cc., which is one of the highest levels, in a case in which the patient recovered, of which we could find record either in the literature or through communication[5,16] with two large centers where the artificial kidney is in use. Berman and co-workers[2] recently reported phenobarbital content of 25.6 mg. per 100 cc. in a patient treated successfully with hemodialysis.

REPORT OF A CASE

A 50-year-old white woman was admitted to hospital November 13, 1950, after having taken approximately 3.5 gm. of phenobarbital. The blood content of the drug was 11.2 mg. per 100 cc. on admittance. Within 12 hours it had dropped to 10.6 mg. per 100 cc. and by end of 36 hours it was 10.1 mg. per 100 cc. and the patient then responded to pain. She was treated at first with caffeine sodium benzoate, picrotoxin and Metrazol (pentylenetetrazol) intravenously. The following day she was given benzedrine, starting with 40 mg. intravenously, then 20 mg. every 30 minutes for nine hours. After nine hours analeptic drugs were discontinued. At the end of 72 hours, the patient was awake but had periods of confusion, and by the fifth day was completely lucid.

She was apparently well until June 24, 1955, when she took approximately 12 grams of phenobarbital. When she was taken to another hospital about eight and a half hours later, gastric lavage was carried out. The total quantity of stomach contents and washings was not measured, but the concentration of phenobarbital in a specimen of the material was 19.3 mg. per 100 cc.

The patient was seen in the emergency room of our hospital when transferred there approximately ten hours after ingestion of the drug. The blood pressure then was 100/60 mm. of mercury and the pulse rate 100. The pupils were constricted and did not react to light. The gag, corneal and deep tendon reflexes were absent. There were scattered ronchi throughout both lung fields. The patient received 3.25 mg. of caffeine sodium benzoate, one ampule (1.5 cc.) of Coramine® (nikethamide) and 50 mg. of amphetamine, without noticeable effect. An oral airway was inserted and suction was applied frequently. Oxygen by nasal catheter was begun and an indwelling urethral catheter was inserted. Because of falling blood pressure, administration of Levophed® (levarterenol bitartrate) was begun and the blood pressure then was maintained at satisfactory levels. During the first night, the patient received a total of 21 cc. of picrotoxin and 1.5 cc. of Coramine every half hour for a total of 12 doses without noticeable improvement. On the morning after admission, she received another 21 cc. of picrotoxin in a three-hour period without effect, and it was decided to discontinue the use of all analeptic drugs as the vital signs had remained quite stable on supportive therapy alone. The content of barbiturate in the blood approximately 24 hours after ingestion was 21.6 mg. per 100 cc. Shortly thereafter, edema of the tongue was noted and an endotracheal tube was inserted. On the second morning, the blood pressure remained stable, but respirations were only 11 per minute. The urinary output was satisfactory. The barbiturate content of the blood was then 17.4 mg. per 100 cc. Respirations were deep and regular and, although ronchi were heard over both lung fields, aeration appeared to be

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Submitted June 28, 1957.

adequate. Administration of oxygen was discontinued but cyanosis developed within five minutes and it was resumed.

There was gradual increase in edema of the extremities, face and tongue. The use of an artificial kidney was considered but transporting the patient to it was deemed too hazardous. In view of the decrease in the barbiturate blood level and the patient's satisfactory condition, it was felt that conservative management was indicated. At 8 p.m., on June 26, respiration suddenly became very shallow and very slow. The patient was placed in a tank respirator and respirations were maintained at approximately 16 per minute. Tidal air flow was measured frequently by the vitalometer and arterial oxygenation by the ear oximeter. These instruments indicated the effectiveness of the respirator and oxygen therapy. By noon on June 27 the patient had a rectal temperature of 103°F. and frequent suction by way of the endotracheal tube was necessary. Oxygen and Levophed were administered continuously. On June 28 transient knee jerks were present bilaterally. The intercostal muscles began to resume functioning to aid in respiration and the patient was removed from the respirator. The blood level of barbiturate at this time was 15.3 mg. per 100 cc. At about noon on June 28 it was noted that there was decreased excursion of the right lung. Atelectasis was suspected and x-ray examination confirmed the suspicion.

The patient was examined by the chief of thoracic surgery and the chief of anesthesiology, who felt that meticulous tracheal toilet now would suffice although they stressed the possibility of an early need for tracheotomy and bronchoscopy. Replaced in the respirator, the patient improved somewhat, but as the respiratory exchange, measured by the vitalometer, was better when she was out of the respirator, she was removed again. Levophed was discontinued, as the blood pressure had stabilized within normal limits.

Inasmuch as the use of the artificial kidney would require transportation of the patient to another hospital, which appeared unwise, peritoneal dialysis was planned for the morning of June 29. At approximately 7 o'clock that morning, however, the patient had a pronounced decrease in ventilatory excursion and cyanosis, requiring her return to the respirator. No air exchange through the endotracheal tube was detected. Tracheotomy was performed, with prompt return of good respiration, aeration of the right chest and improvement of color. Upon examination the endotracheal tube was seen to have become completely blocked with thick, inspissated mucus. This blockage occurred in spite of frequent suctioning and cleansing of the tube while in place. Respiratory exchange was still below normal, and oxygenation, as measured by the oximeter, was also diminished. There was minimal aeration of the left lung. Bronchoscopy was performed and much thick mucoid material was removed from the left bronchus.

Thereafter the patient had no respiratory difficulty. The barbiturate content of the blood was 10.9 mg. per 100 cc. on June 29, and the next day knee jerks and the triceps reflex returned on the right side. On July 1 a gastric tube was passed and the patient was fed through it. On a high protein formula, diarrhea developed, but it improved when the diet was changed to skim milk and broth. By July 2 the patient had biceps reflexes bilaterally and the barbiturate level was 3.7 mg. per 100 cc. of blood. By 1 p.m. that day she responded to painful stimuli. During the day, the patient was noted to have cardiac arrhythmia; an electrocardiogram showed atrial premature beats with aberrant conduction, and there were some nonconducted beats. No treatment was given for the arrhythmia, and within 24 hours it improved. On July 3 the tracheotomy tube was taped and the patient was able to talk. She remained groggy and had occasional periods of confusion until July 6, which was 13 days after ingestion of the barbiturate. At that time it was felt she had completely recovered from the effects of the barbiturate and the drugs given therapeutically. This was 13 days after initial intoxication. The patient was then referred to the Neuropsychiatric Clinic where she was followed on an outpatient basis.

DISCUSSION

The barbiturates are an important group of compounds which are mainly used for depressant action. New compounds are constantly being introduced, but in general, they can be divided into long, medium, short and ultra-short acting drugs. Although they may be so classified, the duration of action depends upon dosage, individual response and routes of administration, as well as the particular drug used.

Soon after absorption there is a relatively homogenous distribution throughout the body, but a slightly greater concentration in the liver and kidneys than elsewhere. What becomes of a particular barbiturate within the body evidently depends on its chemical structure, but one cannot predict from chemical structure exactly how the body will deal with the drug. In general, however, the thiobarbiturates and those with complex cyclic radicles, such as hexobarbital, are less stable and are almost completely destroyed in the liver. Barbiturates with short alkyl radicles, such as phenobarbital, are more likely to be stable and are excreted in the urine.[8]

The primary barbiturate effect is that of depression of the central nervous system. Two acceptable theories as to the means of its action are: (1) Decreased response of the cerebral cortex to sensory stimuli and (2) depression of the sleep center located in the diencephalon. The latter theory, which is the more widely accepted, must presuppose that the sleep center is more sensitive to the drug, for it has been shown that the drug is relatively uniformly distributed throughout the brain. Toxic effects are primarily exaggerations of the normal response.

With large doses, depression of respiration ensues, which is the usual cause of death.[6]

The length and depth of coma is better correlated with the content of drug in the blood than with the amount ingested. Sunshine and Mackett[18] divided a series of cases clinically into five groups of varying degrees of depression, noting blood level determinations as the patients passed from stage to stage.

The blood level and the type of drug determine the clinical state, and in general the longer-acting group requires higher blood levels to produce the varying levels of depression. In cases of phenobarbital poisoning, one can fairly well predict the clinical course by the blood barbiturate level. Levels of more than 10 mg. per 100 cc. of long-acting barbiturates, 7 mg. per 100 cc. of intermediate-acting, and 3 mg. per 100 cc. of short-acting barbiturates can cause death.[3] The presence of such amounts, however, does not constitute prima facie evidence of death from barbiturate poisoning.

Treatment of overdoses of barbiturate varies widely in various clinics. We consider nearly all forms of therapy at one time or another during the treatment of patients.

The two main schools of treatment are that of Koppanyi and Fazekas,[10] who advocate analeptic drugs in large doses, and that of Nilsson,[13] who expressed belief that supportive measures are sufficient. Certain basic nursing and medical procedures must be carried out in all cases. These include maintenance of a patent airway, maintenance of blood pressure and nutrition, prevention of trophic skin changes and prevention of infection. The time-honored analeptic and stimulatory drugs are usually thought of. (Use of them was begun in the case herein reported.) Many drugs have been tried, but the two most frequently relied on are pentylenetetrazol and picrotoxin.[10,14]

Some investigators believe that picrotoxin and pentylenetetrazol should be the mainstays of treatment because they act upon the entire central nervous system and physiologically reduce the depressing effect of the barbiturates.

Pentylenetetrazol is a central nervous system stimulant acting upon the medulla and spinal cord. Bailey, Goth and Lackey[1] studied the effect of the drug on dogs. Trying various combinations of doses of pentylenetetrazol and barbiturate after a known toxic level was established, they noted that pentylenetetrazol did not protect or resuscitate the animals. Nor did it maintain the blood pressure; in fact, depressed it. Picrotoxin also has an early stimulating effect on the central nervous system of subjects with barbiturate intoxication; but depression ensues, and death due to respiratory failure may follow.

Several factors make it difficult to evaluate the effectiveness of analeptic drugs in the treatment of barbiturate intoxication. These include: Status of renal function, visceral pathologic changes, gastric lavage (which may reduce the amount of barbiturate), the time interval between the overdose of barbiturate and the beginning of treatment, synergistic action of more than one depressant, and overstimulation with the analeptic drug.[7]

In 1955, Shaw[17] in Australia reported upon the use of a barbiturate antagonist, "Megimide," for lessening respiratory depression and shortening coma, but this drug was not available for use in the case herein reported.

Baker and Michael[12] reported very good results obtained with an electric stimulater in 14 of 15 patients who were in coma from overdose of barbiturates. They did not say, however, the amounts of barbiturates ingested or the amounts in the blood. The method has not been in use long enough to evaluate the results obtained. It does not increase the metabolism or excretion of the drug.

Had we had an artificial kidney available, much time and effort might have been saved in the case herein reported. Kyle[11] pointed out that the artificial kidney can remove the offending drug 20 to 30 times as rapidly as it is excreted in the urine. Berman and co-workers[2] expressed belief that hemodialysis should be used in two basic situations: When the amount of drug ingested or the blood barbiturate level is in the potentially fatal range; and when the underlying physical state is likely to increase the hazards of coma. In the case herein reported, it was not felt necessary at first to transfer the patient to a hospital that had an artificial kidney, since there was an apparent satisfactory drop in the blood level and on a previous occasion she had recovered rapidly despite a rather high blood level of barbiturate; and then, when it became apparent that she was not responding well, her condition was such that transfer was considered too hazardous.

Peritoneal dialysis was also considered, but was not feasible because by then the patient had to be kept in a tank respirator. Reporting on the use of peritoneal dialysis in three patients Henninger[9] noted an increase of between 1.5 and 2 gm. removed in four days by this method.

A conservative method of treatment advocated by Nilsson,[13] Clemmesen[4] and others embraces the following features: Aspiration of stomach contents, maintenance of a free airway, use of antibiotics, antishock therapy, making certain of diuresis, administration of vitamin C in prolonged coma because of increased vascular permeability, use of extra bed clothes if hypothermia occurs, use of cold packs if hyperthermia is a factor, daily routine nursing care around the clock.

Nilsson[13] expressed the opinion that stomach aspiration should be performed in only the first hour or two after ingestion of barbiturate and should not even be attempted after four hours. He and other investigators have warned against lavage because of the possibility of aspiration of fluid. Other observers, however, including Reed,[14] have expressed belief that gastric lavage should be performed regardless of the time interval, for a certain amount

of irritation of the stomach is caused by large doses of barbiturates, which may cause pylorospasm and gastric retention of the drug for many hours. It is pertinent that in the present case when gastric lavage was carried out, eight and one-half hours after ingestion of phenobarbital, the amount of the drug in the fluid was 19.3 mg. per 100 cc.

Vital to any method of treatment is the maintenance of an adequate airway. Lack of strict and constant vigilance as to proper bronchial toilet and pulmonary ventilation is often the cause of an unfavorable outcome. Tracheotomy should be considered early if there are signs of difficulty with retained secretions. In the present case tracheotomy probably should have been done soon after the endotracheal tube was inserted. This procedure might have prevented the development of pulmonary atelectasis and the need for bronchoscopy.

Clemmesen in Denmark[4] observed that the addition of antishock therapy, continuous use of oxygen, centralization of treatment in one hospital, and abandonment of all stimulation caused a drop in the mortality rate in cases of barbiturate intoxication from 25.4 per cent to 3.7 per cent.

SUMMARY

A case of barbiturate intoxication with prolonged coma and high barbiturate blood level with survival is presented. Treatment was mainly supportive, with strict attention to the maintenance of vital functions. A brief presentation of the pharmacologic features of barbiturates and several methods of treatment are discussed.

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California MEDICINE

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EDITORIAL

Physicians and Lawyers

ON ANOTHER PAGE of this issue appears an article prepared jointly by the chairmen of liaison committees of the California Medical Association and the State Bar of California. This item outlines the agreement which has been reached by the respective committees of the two organizations on the single subject of expert medical witness for medical malpractice plaintiffs.

While some members of the medical profession—and doubtless many of the legal profession—may raise their eyebrows over the advent of such an agreement, there appears ample good reason why this step has been taken.

Over the years physicians and lawyers have met in the courts of the land. Where physicians appear as witnesses on opposite sides of a case and present testimony at variance with each other's, they may be pictured by attorneys, judges and jurors as something less than upright men pursuing the same scientific course. At times the entire medical profession is castigated as being composed of men who would take sides to the exclusion of their scientific training.

In many such suits physicians have found themselves badgered by opposing counsel, sometimes faced with the reading of sections taken out of context from medical books. Such situations have been embarrassing to the physician witness and derogatory to the whole profession of medicine, to say nothing of the effect on the course of justice.

The very appearance of these situations in our courts has caused many of the meditative minds in both professions to seek an avenue of approach to a problem which pits one professional group against another, to the benefit of neither, to the detriment of both and the disservice of equity. A baiting attorney, even though using his tactics simply as a technique, must leave the impression of a bully on at least some jurors and courtroom observers. A faltering physician, even though medically sound in his testimony, can easily create the impression that medicine has not progressed to a very high scientific level.

That some progress has been made in working for something better has become apparent in recent years. In several states and in numerous counties throughout the country medical-legal groups have sat down together for frank discussions of their joint problems and have come up with some of the answers.

The result of these joint sessions in many places has been the adoption of an interprofessional code of conduct. Such items as the conduct of both lawyer and physician while the latter is on a witness stand have been spelled out in some of these codes. The subpoenaing of physicians' records; the calling of physicians as expert but unwilling witnesses for purposes of picking their brains; the relationship of physician and attorney on both sides of a case—these and other details have been analyzed and reduced to rules of conduct which both professional groups understand and accept.

The adoption of these codes represents a healthy and progressive movement on the part of both groups. It is certain that much misunderstanding and hard feelings will be eliminated and the dignity of both enhanced by these agreements.

More recently there has come another indication of better and closer interprofessional cooperation. In one large state a program for the naming of physician experts to give impartial medical testimony in personal injury cases has been developed. These experts are chosen from panels by the judges

and their main function is to give impartial testimony without any thought of bias for or against a plaintiff or defendant. While this process has not spread very wide to date, it is being carefully observed in many other areas. If it produces the results hoped for, the bickering between expert witnesses in highly contested cases may be eliminated; in fact, many cases may be settled before trial if the impartial medical testimony will not support the legal claims made.

The newest move in California comes as a follow-up of these earlier evidences of collaboration between the two professions. The maintenance of panels of experts for the use of plaintiffs' attorneys in professional liability cases should go a long way toward dispelling the impression which has been voiced by some lawyers and judges that a "conspiracy of silence" exists in the medical profession when a medical malpractice case comes to court. Where panels of medical experts are established to work under the guidance of both the medical and the bar associations, there would be little to sustain this sort of reasoning. The program has been agreed upon by both parties under a set of rules which will protect all parties against abuses and still make expert testimony available to the plaintiff.

Where large county medical societies are involved, it is expected that the county society will supervise the program through its own staff. In smaller counties, area panels will be developed to work with the cooperation of the district councilor of the California Medical Association. In either event, the medical societies will be advised on all cases and adequate administrative controls will be maintained. [Details are given in the article appearing on page 173 of this issue.]

While only a limited experience has been had in this program to date, it appears obvious that the plan will be put into effect statewide at the earliest possible date and that the medical and legal professions can take another step forward on the path to mutual respect and understanding. But above all, the public will be presented a service which could not be purchased for money but can be extended with good will by the two large professions involved. This is at once good citizenship and good public relations at its very best.

Editorial Comment . . .

Why a Tumor Registry?

THE QUESTION is often asked, "Why should a hospital have a tumor registry?" The administrator questions the cost and asks how such a charge can be justified in terms of service to all the patients cared for in the hospital. The staff member is concerned in what he receives in return for the time spent in obtaining follow-up information since the family physician, to whom the patient is returned for further care, resents the "specialist" periodically asking for information relative to the condition of the patient.

One answer might be that, cancer being a multiplicity of diseases, every method that may provide any information should be developed. There are those who state that the collection of such data for statistical analysis is time consuming and futile, that statistics never cured a case, that all the patients die sooner or later in spite of treatment and perhaps because of treatment. Others, the optimistic ones, say that cancer can be cured in 85 per cent of cases if treated early enough. Between these two groups are the careful scientific members of the profession who are interested in the results of their therapy not only at the time of discharge of the patient from the hospital but in the years that the patient lives. It is for this group that a cancer registry becomes a valuable tool in treatment and in teaching.

Few persons are endowed with "total recall" memory, and physicians are not especially favored. They, like others, are likely to remember their successes and forget their failures and particularly the details of the cases with the passage of time. Hence the need of accurate recording of the facts as they develop. Such a clinical record becomes quite bulky, and to review all such records for information as to age, sex, site, type, diagnosis and therapy (or reason for no therapy) is time-consuming.

A tumor registry is designed to expedite and make attractive such reviews. Pertinent information is abstracted and filed on all patients with cancer seen in the hospital departments. For the convenience of the specialists the abstracts may be filed according to site of lesion. The follow-up informa-

tion is kept with the abstract and thus a running inventory is maintained. A yearly audit is easily prepared and the staff made aware of the cancer program of the hospital.

The obligation of sharing the knowledge gained through experience is traditional with the medical profession. The means of sharing such knowledge is the presentation of data before medical societies or specialist groups and by writing for the professional journals. The tumor registry provides the basic material for such writings. True, it will be necessary in certain cases to review some clinical records individually for specific details not recorded in the abstracts, but the identification of special cases has been made easier. Since the end results are *sine qua non* evaluating any information, complete follow-up data is essential.

Nothing has been said of the standardization of report forms and of the terms used. So long as the hospital is operating the registry as an individual program, standardization is of small consequence; but when the hospital becomes one of many participating in a central tumor registry, standardization becomes necessary to insure that all participants use the same terms to define or describe the same material.

Some persons may ask why a central tumor registry is necessary, particularly if all hospitals are maintaining a complete tumor registry? The answer is that relatively few hospitals record more than five hundred cases of cancer a year; most of them have about two hundred cases a year. These cases include all sites, including the skin. In some institutions malignant lesions of certain sites are seen rarely. It becomes necessary, then, that all such experiences be pooled in order that a better knowledge of these "rare" lesions may be gained and better diagnostic and therapeutic techniques developed. A central tumor registry would also provide means for evaluating the efficiency of therapeutic techniques by the statistical analysis of the effects upon the well-being of the patient and the length of survival, using the accepted techniques for a base.

As Dr. Harvey Cushing once said: "Records based on observation have an enduring value that transcends all our social philosophies; and the doer always does over again—always a little better, we would like to believe—what someone has done before and someone before *him*."

JAMES W. ELLIS, M.D.
Cancer Consultant to the California State Department of Public Health

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Important!

SCIENTIFIC MOTION PICTURES

Attention—All Members of the California Medical Association

AT THE 1958 annual meeting of the California Medical Association, to be held at the Ambassador Hotel, Los Angeles, April 27 through April 30, approximately 100 motion pictures on surgery and medicine will be shown on the program of the Motion Picture Division.

The educational and scientific value of motion pictures has been proven by the large attendance at the Motion Picture Section during our previous meetings.

If you would like to present a scientific motion picture film on this program, you must submit your application to the committee for approval not later than February 15, 1958.

Address all communications to: Arthur E. Smith, M.D., D.D.S., chairman, Motion Picture Division, California Medical Association, 1930 Wilshire Boulevard, Suite 511, Los Angeles 57, California.

California MEDICAL ASSOCIATION

NOTICES & REPORTS

Medical Expert Panels for Malpractice Cases

IN RELATIVELY recent years there has been an appreciable increase of interest in attempts to foster better understanding and closer cooperation between the medical and legal profession.* It is the considered judgment of many that lack of understanding or cooperation arises most frequently in the exceptional cases wherein the respective roles of the lawyer, client-patient and doctor are not understood by one or more of these three participants. Regardless of the causes for the problems which have arisen, the California Medical Association and the State Bar of California simultaneously concluded to investigate and confer on the general subject and initially on the availability of impartial medical expert witnesses in alleged medical malpractice cases.

At its 1955 meeting the Conference of State Bar Delegates adopted Resolution No. 39 which provided for the study of the problem of obtaining evidence in malpractice cases. As a result of that resolution a committee was appointed to make that study.† That committee investigated the problems individually, conferred together and with representatives of C.M.A. In August 1956, the committee submitted its report, recommending that the Executive Committee of the Conference of State Bar Delegates or the Board of Governors appoint a committee to confer with a comparable committee to be appointed by the Council of the California Medical Association and if possible to formulate a plan to alleviate the misunderstandings and establish closer cooperation between the two professions. In March of 1957, the President of the State Bar, pursuant to authority of the Board of Governors, appointed a committee to confer with a committee appointed by the Council of C.M.A.‡ After several conferences the joint liaison committees formulated a plan as follows:

1. *Purpose:*

A panel of physicians available for consultation with attorneys of the local or neighboring bar associations for pre-suit expert opinion and, where suit is instituted, for testimony as an expert witness.

Such panel to be used *only* for cases of alleged medical malpractice.

2. *Selection of Panels:*

On appointment basis by the medical societies within each territorial district with approval of panel by the board of directors of the applicable local county bar association.

3. *Disqualifications of Panel Members:*

If panel physician is treating or has ever treated or consulted upon the plaintiff patient; or if there is a relationship, business or otherwise, between panel and defendant physician.

4. *Make-up of Panels:*

a. General practice.

b. The following basic specialty areas of medical

*For example, see Medical Testimony: Doctors and Lawyers Cooperate, by Audrey Barr, *Journal of the American Judicature Society*, Volume 41, Number 3, dated October 1957, page 78, and *Journal of the American Medical Association*, Volume 164, August 24, 1956, page 1931.

†Donald A. Odell, Sr., Los Angeles, chairman; R. Winfield Achor, Santa Rosa; Fitz-Gerald Ames, Sr., San Francisco; James W. Archer, San Diego; Scott L. Harrington, San Francisco; Thomas E. Heffernan, Corona Del Mar; Daniel J. Higgins, Auburn; Willard Lee Pope, Watsonville; John B. Rosson, Oakland; Alfred Thomas, Fresno; Will H. Winston, Long Beach. Liaison: John S. Frazer, Los Angeles; Edward W. Schramm, Santa Barbara.

‡*C.M.A. Committee:* Joseph F. Sadusk, Jr., M.D., Oakland, chairman; Donald A. Charnock, M.D., Los Angeles; Rees B. Rees, M.D., San Francisco; Donald E. Ross, M.D., Los Angeles; Francis E. West, M.D., San Diego.

State Bar Committee: Donald A. Odell, Los Angeles, chairman; James Archer, San Diego; John Finger, San Francisco; Dennis Hession, San Mateo; Elwood Murphey, Oakland.

FRANK A. MacDONALD, M.D. . . . . President
FRANCIS E. WEST, M.D. . . . . President-Elect
JAMES C. DOYLE, M.D. . . . . Speaker
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JOHN HUNTON . . . . . Executive Secretary
General Office, 450 Sutter Street, San Francisco 8
ED CLANCY . . . . . Director of Public Relations
Southern California Office:
2975 Wilshire Boulevard, Los Angeles 5 • DUnkirk 5-2341

practice, and such additional areas of specialty practice as shall be determined within each district: (1) Medicine, (2) surgery, (3) obstetrics and gynecology, (4) pediatrics, (5) pathology, (6) radiology.

5. *Administration of Panel:*

By a designated officer of the local county bar association. Names on file.

6. *Method of Call of a Panel Member:*

By complainant's request in writing to a physician on a panel list as submitted by local bar association. The physician shall notify his county medical society and the local bar association in writing of the requested consultation, giving name of attorney, complainant and physician complained against.

7. *Education of Panel:*

By a series of lectures from a joint committee of local medical and bar associations; or by special statewide indoctrination committees.

8. *Reimbursement for Consultation or Testimony:*

For the consultation and examination, review of records, preparation of report, and testimony, the physician shall receive the reasonable value of his time in accordance with the prevailing fees in his community.

9. *Panel Physicians in Court:*

No reference shall be made in court to the panel or medical or bar associations' participation therein. Judges shall be requested to adopt a rule forbidding counsel (on each side) to mention status of witness in relation to panel.

10. *Availability of Panel Members to Defense:*

a. A panel physician who consults with or examines a complainant may not, without the consent of the complainant, be examined in a civil action as to any information acquired from the complainant which was necessary to enable the physician to prescribe or act for the complainant, except as provided below.

b. A complainant may secure additional examinations or consultations from the panel list, but only if the complainant agrees in writing with the preceding panelist consulted that the latter may be examined in any civil action based upon the complaints involved.

c. A complainant may secure an examination or consultation with a second member of the over-all panel without being required to waive the confidential nature of the first examination or consultation providing the initial panelist selected reports in writing to the administrative officer of the local county bar association that the complaint is not within his area of practice; or that a second examination or consultation is necessary or advisable for stated cause.

TERRITORIAL DISTRICTS

1. The district organizations for panels of experts on a regional basis shall be coextensive with the councilor districts of the California Medical Association, except that the third and fourth councilor districts (both in Los Angeles County) shall be combined to function as one district. Each district shall be asked through its California Medical Association councilor to supervise the establishment of a panel.

2. Lawyers shall use the panel within their own territorial district and not elsewhere and physicians on the panels shall be expected to respond to requests from within their own councilor district and not from elsewhere.

3. Inasmuch as bar associations do not extend beyond county lines, as do some of the rural medical societies, and inasmuch as there is more than one bar association in some counties, each county bar association shall be requested to set up the appropriate administrative machinery to administer the program within its geographical area on behalf of all attorneys in the county regardless of the bar association to which they belong, and to establish such liaison with other bar associations as each county may determine desirable; lawyers in counties where there is no bar association shall be given an opportunity to join with one adjacent county where there is a bar association for purposes of administration of the panels.

At their September 1957 meetings the Council of the California Medical Association and the Board of Governors of the State Bar of California accepted and approved this plan and subsequently appointed new committees charged with the responsibility of implementing and endeavoring to make effective the plan.* As a part of its resolution of approval, the Board of Governors further requested the committee of the State Bar to consider the necessary steps to procure by rule or statutory amendment implementation of Rule 9 (re reference in court to the panel) and Rule 10 (re availability of panel members to the defense).

The joint liaison committees met again on December 12, 1957. Also present and participating most helpfully in the deliberations were Edwin A. Heafey, Esq., president of the State Bar, Howard Hassard, Esq., counsel for C.M.A. and Rollen Waterson, executive secretary to the C.M.A. committee.

**C.M.A. Committee:* Francis E. West, M.D., chairman; Donald E. Ross, M.D.; C. J. Attwood, M.D., Oakland; Rees B. Rees, M.D.; Donald A Charnock, M.D.

State Bar Committee: James Archer, chairman; Frank S. Balthis, Los Angeles; Dennis Hession, John Finger, Elwood Murphey.

Subject to approval by the governing bodies of the two organizations, the joint liaison committees recommended that the President of C.M.A. and the President of the State Bar request that similar joint liaison committees be appointed by the councilor of each district of C.M.A. and the presidents of each county bar association to implement the plan on the local level. It is recommended that members of the local committees be urged to confer and work with the members of the state-wide liaison committees in order that there may be as much uniformity in the program as is practicable considering the special problems that may exist in certain areas. It is anticipated that designated personnel of C.M.A. will call upon C.M.A. district councilors and the appropriate members of the county bar committees to assist in initiating the program. It is further recommended that the executive secretaries of the county medical societies be charged with the responsibility for the administration of the plan in each area. The reason for this being that in most areas the medical societies maintain permanent offices and staffs. Conversely, comparatively few county bar associations are similarly established. Therefore, it was recommended that administration of the panel list should not be delegated to a secretary but should become the duty of one of the officers of the county bar associations.

The members of the panel will be fully indoctrinated as to their role, responsibilities and procedures. Members of the county bar associations' committees shall participate in the indoctrination lectures. More detailed information regarding the indoctrination will be made available in the near future in the form of an "Indoctrination Kit." Certain forms to assure proper controls and desired statistical information were recommended by the joint committees. They will be made available to each county medical society.

Certain specific "ground rules" are recommended by the joint committees, subject to approval of the two governing bodies. They are as follows:

1. Panel members are to be appointed by the county society, not selected from volunteers.

2. Only those panel appointees who have attended the indoctrination lectures are eligible to serve.

3. Present members of public service or malpractice committees of county societies are not eligible for service on the panel.

4. Panel appointments are for one year only; a new panel is appointed each year, but the same physicians are eligible year after year.

5. Panel members, as such, are available to complainants against doctors of medicine only.

6. In each county, the panel should be representative of the practice of medicine in that area as to specialties and general practitioners.

7. The financial arrangements between the complainant's attorney and the panel member are to be by agreement between them, although a fee scale may be established by the county medical society if it so desires. Fees are to be paid in advance unless otherwise agreed between the panel member and the attorney.

Individual county medical societies and county bar associations are free, of course, to modify this proposed plan to meet local conditions and attitudes; in fact, they are free to accept or to reject the entire concept of the proposed establishment of professional liability medical panels.

It was recommended by the joint liaison committees that Rule 9 of the proposal should be implemented by the Board of Bar Governors through the Committee on Administration of Justice. However, the committees proposed a rule as follows:

"No reference shall be made in any action or proceeding to the existence of a joint county medical-bar association panel of physician experts or to the participation or nonparticipation of any witness therein."

It was the consensus of the State Bar Committee that no action is required for the implementation of Rule 10 as the participation of the physicians is voluntary and if a lawyer does not desire to adhere to the program, he can be denied the use of the panel.

As this is a new and unique program, it will take some time and planning before it can be inaugurated in some areas and before it can be tested effectively. However, there has been a somewhat similar plan in operation for several years in Los Angeles County and the experiences there were very beneficial to the joint liaison committees and should be of benefit in other localities. It is the sincere hope of all interested in the plan that it succeed and tend substantially to alleviate some of the misunderstandings that seem to presently exist.

FRANCIS E. WEST, M.D.
President-Elect of California Medical Association

JAMES W. ARCHER
Chairman of State Bar Liaison Committee

CANCER BOOKLETS for *Your Patient*

THE CANCER COMMISSION and the California Division of the American Cancer Society wish to remind physicians of booklets and leaflets on cancer which are available without cost for distribution to patients. The busy physician will find these educational materials helpful to supplement his advice to patients. The following are recommended:

GIVE YOUR DOCTOR A CHANCE... This booklet describes the general nature of cancer, stresses the importance of annual checkups, and explains how the family physician makes a cancer detection examination.

PERSONAL MEMO... This leaflet explains the importance of routine breast self-examination and illustrates how a woman can carry out this technique.

TO SMOKE OR NOT TO SMOKE... This booklet reviews the research studies on this subject and states the position of the American Cancer Society.

CANCER FACTS FOR MEN...

CANCER FACTS FOR WOMEN... These companion leaflets present basic information in concise fashion.

101 ANSWERS TO YOUR QUESTIONS ABOUT CANCER... A 20-page booklet answering the questions most frequently asked about cancer.

WILL A CANCER QUACK GET YOU?... This booklet gives a brief message on the cancer quack problem.

-------- USE THIS ORDER BLANK --------

AMERICAN CANCER SOCIETY
(California Division)
467 O'Farrell St., San Francisco

Please send me the indicated quantities of the following:

..... Give Your Doctor a Chance
..... Personal Memo
..... To Smoke or Not to Smoke
..... Cancer Facts for Men
..... Cancer Facts for Women
..... 101 Answers
..... Will a Cancer Quack Get You?

Name.....

Address.....

.....

Standards for Blood Bank Sponsorship By the California Medical Association*

1. Blood banks will operate on a nonprofit principle. A copy of the letter of exemption from the United States Bureau of Internal Revenue and the State Franchise Tax Board will be submitted. A letter will be submitted annually by the blood bank president or other responsible officer stating that the blood bank is complying with its nonprofit status. A letter will be submitted annually from the blood bank auditor stating that blood bank funds are not being used for purposes other than that for which the blood bank was formed and that during the past year there have been no substantive changes in the articles of incorporation or the by-laws of the blood bank, except as follows:

2. Blood banks will perform an acceptable community service. Adequate amounts of all types of blood will be supplied to all qualified medical facilities requesting service in the geographic area covered by the individual blood bank at minimum cost to the patient. The method by which this is accomplished is the responsibility of the individual blood bank as long as the basic effort for blood procurement follows the replacement principle.

3. Blood banks will fulfill basic technical standards as outlined by the National Institute of Health and/or the California State Department of Public Health.

4. Each blood bank will be sponsored by the county medical society where it is located. Evidence of such sponsorship will be submitted annually to the C.M.A.

5. The above information will be secured by the C.M.A. Committee on Blood Banks and will be submitted annually to the Council of the C.M.A.

*Approved by the Council, January 11, 1958.

Stolen Child

(Bulletin from the San Francisco Police Department)

ON FRIDAY, December 20, 1957, the following described infant was stolen from 519 Fell Street in San Francisco:

Baby Caroline Elizabeth Horton, 11 weeks old, 11 pounds, 12 ounces, 23¼ inches long, curly thick black hair with deep cowlicks, real thick and long sideburns that partially obscure a small birthmark on right side of face, large brown eyes, light brown complexion.

This baby was born with six fingers on both hands and as these were amputated shortly after birth, the amputation marks are still visible on sides of both little fingers. The mark on the left little

finger is a noticeable small dark spot and the mark on the right little finger is a small nub.

When the baby was stolen it was dressed in a white dress, yellow slip, diapers, yellow ruffled rubber panties, white shoes, yellow socks, yellow ribbon in hair, yellow sweater with pink flowers on side, yellow corduroy bonnet trimmed in white, yellow nylon blanket.

Anyone having any information on the above subject, please contact the General Work Detail of the San Francisco Police Department, SU. 1-2020, Extension 411.

In Memoriam

BERNSTEIN, THEODORE I. Died in Los Angeles, December 11, 1957, aged 48, of leukemia. Graduate of the University of Southern California School of Medicine, Los Angeles, 1938. Licensed in California in 1938. Doctor Bernstein was a member of the Santa Barbara County Medical Society.

✤

DONALD, WILLIAM GOODRICKE. Died in Berkeley, December 30, 1957, aged 68, of acute myocardial infarction, coronary sclerosis. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1923. Licensed in California in 1923. Doctor Donald was a member of the Alameda-Contra Costa Medical Association.

✤

HARRISON, WILLIAM H. Died December 10, 1957, aged 73, of heart disease. Graduate of the College of Physicians and Surgeons of San Francisco, 1907. Licensed in California in 1938. Doctor Harrison was a member of the San Francisco Medical Society.

✤

HARSHA, WILLIAM T., JR. Died in Pasadena, November 28, 1957, aged 49. Graduate of Stritch School of Medicine of Loyola University, Chicago, Illinois, 1935. Licensed in California in 1944. Doctor Harsha was a member of the Los Angeles County Medical Association.

✤

HARTMAN, GEORGE W. Died in San Francisco, December 24, 1957, aged 78. Graduate of Johns Hopkins University School of Medicine, Baltimore, Maryland, 1908. Licensed in California in 1909. Doctor Hartman was a retired member of the San Francisco Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✤

KELLEY, DOUGLAS M. Died in Berkeley, January 1, 1958, aged 45, of anoxia, cyanide poisoning. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1937. Licensed in California in 1937. Doctor Kelley was a member of the Alameda-Contra Costa Medical Association.

✤

KLEMME, ROLAND M. Died in Salinas, November 21, 1957, aged 61. Graduate of Washington University School of Medicine, St. Louis, Missouri, 1921. Licensed in California in 1950. Doctor Klemme was a member of the Monterey County Medical Society.

✤

NICHOLSON, JAMES W. Died in Porterville, December 4, 1957, aged 70, of metastatic carcinoma. Graduate of Vanderbilt University School of Medicine, Nashville, Tennessee, 1913. Licensed in California in 1913. Doctor Nicholson was a member of the Tulare County Medical Society.

✤

OSTER, EDWIN A. Died in Campbell, December 20, 1957, aged 34. Graduate of the University of Illinois College of Medicine, Chicago, 1950. Licensed in California in 1951. Doctor Oster was a member of the Santa Clara County Medical Society.

✤

REDEWILL, FRANCIS H., SR. Died in Whittier, December 20, 1957, aged 78, of uremic poisoning. Graduate of Johns Hopkins University School of Medicine, Baltimore, Maryland, 1906. Licensed in California in 1923. Doctor Redewill was a retired member of the San Francisco Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✤

VOGEL, ESTHER E. Died in Glendale, December 13, 1957, aged 44, of ovarian carcinoma with metastasis. Graduate of the College of Medical Evangelists, Loma Linda-Los Angeles, 1939. Licensed in California in 1939. Doctor Vogel was a member of the Los Angeles County Medical Association.

✤

VRUWINK, JOHN. Died in Pasadena, November 18, 1957, aged 67. Graduate of Rush Medical College, Chicago, 1916. Licensed in California in 1917. Doctor Vruwink was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

✤

WAYLAND, RAYMOND T. Died in San Jose, December 16, 1957, aged 67, of stroke. Graduate of Jefferson Medical College of Philadelphia, Pennsylvania, 1913. Licensed in California in 1915. Doctor Wayland was a member of the Santa Clara County Medical Society.

✤

YOUNG, CHARLES S. Died in Los Angeles, December 14, 1957, aged 65, of pulmonary fibrosis. Graduate of the College of Physicians and Surgeons, Los Angeles, 1914. Licensed in California in 1914. Doctor Young was a member of the Los Angeles County Medical Association.

APPLICATION FOR HOUSING ACCOMMODATIONS

FOR YOUR CONVENIENCE in making hotel reservations for the coming meeting of the **California Medical Association,** April 27 to 30, 1958, Los Angeles, hotels and their rates are at the right. Use the form at the bottom of this page, indicating your first and second choice. Because of the limited number of single rooms available, you will stand a much better chance of securing accommodations of your choice if your request calls for rooms to be occupied by two or more persons. **All requests for reservations must give definite date and hour of arrival as well as definite date and approximate hour of departure; also names and addresses of all occupants of hotel rooms must be included.**

ALL RESERVATIONS MUST BE RECEIVED BEFORE: APRIL 1, 1958

Eighty-seventh Annual Session
CALIFORNIA MEDICAL ASSOCIATION
Los Angeles, California
APRIL 27-30, 1958

HOTEL ROOM RATES *

	Single	Twin Beds	Suites
AMBASSADOR HOTEL			
3400 Wilshire Boulevard			
Main Building	9.00-19.00	12.00-22.00	28.00-38.00
Garden Studios	17.00-23.00	22.00-30.00	40.00-54.00
CHAPMAN PARK HOTEL			
3405 Wilshire Boulevard		12.00-16.00	20.00-25.00
THE GAYLORD HOTEL			
3355 Wilshire Boulevard	8.00	10.50	18.00
HOTEL CHANCELLOR			
3191 West Seventh Street	7.00- 8.00	10.00-12.00	
SHERATON-TOWN HOUSE			
2961 Wilshire Boulevard	11.00-18.00	16.00-23.00	31.00

***The above quoted rates are existing rates but are subject to any change which may be made in the future.**

CALIFORNIA MEDICAL ASSOCIATION
450 Sutter Street—Room 2000
San Francisco 8, California

Please reserve the following accommodations for the 87th Annual Session of the California Medical Association, in Los Angeles April 27 to 30, 1958.

Single Room $..... Twin Bedded Room $.....

Small Suite $..... Large Suite $..... Other Type of Room $.....

First Choice Hotel..... Second Choice Hotel.....

ARRIVING AT HOTEL (date).....Hour:.....A.M.....P.M. } Hotel reservations will be held until

Leaving (date).....Hour:.....A.M.....P.M. } 6:00 P.M., unless otherwise notified

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Therefore, please include the names of both persons for each twin-bedded room requested. Names and addresses of all persons for whom you are requesting reservations and who will occupy the rooms asked for:

.....

.....

.....

.....

Individual Requesting Reservations—Please print or type Delegate?..... Alternate?.....

Name..... County.....

Address..... City and State.....

California Medical Association

1958 Annual Session

AMBASSADOR HOTEL, LOS ANGELES *April 27-30*

THREE POSTGRADUATE COURSES

9:00 a.m.-noon

Liver Diseases—A NINE-HOUR COURSE
U.S.C. SCHOOL OF MEDICINE

Abdominal Pain—A NINE-HOUR COURSE
U.C.L.A. SCHOOL OF MEDICINE

Management of Trauma—A NINE-HOUR COURSE
COLLEGE OF MEDICAL EVANGELISTS

A FEE WILL BE CHARGED FOR THE ABOVE COURSES
(For programs, see following pages)

FOUR DAYS

GENERAL SCIENTIFIC MEETINGS
TECHNICAL EXHIBITS · SCIENTIFIC EXHIBITS
MEDICAL MOTION PICTURES

✶

PRESIDENT'S DINNER DANCE

TUESDAY, APRIL 29
COCOANUT GROVE, AMBASSADOR HOTEL

✶

HOUSE OF DELEGATES

SUNDAY, APRIL 27 AND WEDNESDAY, APRIL 30

REGISTRATION DAILY · 9:00 a.m. to 5:00 p.m. · NO REGISTRATION FEE

Please make hotel room reservations only through C.M.A. office in San Francisco
(Use reservation request form on facing page)

87th Annual Session

CALIFORNIA MEDICAL ASSOCIATION IN COOPERATION WITH THE

College of Medical Evangelists
University of California at Los Angeles and
University of Southern California

Presents . . .

Three New Postgraduate Courses During
C. M. A. Annual Session

Abdominal Pain

Program planned by the University of California at Los Angeles School of Medicine

APRIL 28, 29 and 30

West Venetian Room, Ambassador Hotel

MONDAY, APRIL 28

9:00-9:45 a.m.—**Painful Medical Diseases of the Abdomen**—William E. Molle, M.D.
9:45-10:00 a.m.—Recess.
10:00-10:45 a.m.—**Psychiatric Aspects of Abdominal Pain**—Frank F. Tallman, M.D.
10:45-11:30 a.m.—**Panel Discussion**—William E. Molle, M.D., Sherman M. Mellinkoff, M.D., and Frank F. Tallman, M.D.
11:30-12:00 noon—**Questions.**

TUESDAY, APRIL 29

9:00-9:45 a.m.—**Surgical Causes of Abdominal Pain**—Mark M. Ravitch, M.D.
9:45-10:00 a.m.—**Recess.**
10:00-10:45 a.m.—**Retroperitoneal Causes of Abdominal Pain**—Chester C. Winter, M.D.
10:45-11:30 a.m.—**Panel Discussion**—Chester C. Winter, M.D., Sherman M. Mellinkoff, M.D., and Mark M. Ravitch, M.D.
11:30-12:00 noon—**Questions.**

WEDNESDAY, APRIL 30

9:00-10:30 a.m.—**Systemic Causes of Abdominal Pain**—Sherman M. Mellinkoff, M.D.
10:30-10:45 a.m.—**Recess.**
10:45-11:30 a.m.—**Panel Discussion—Differential Diagnosis of Abdominal Pain**—William E. Molle, M.D., Sherman M. Mellinkoff, M.D., Mark M. Ravitch, M.D., Frank F. Tallman, M.D., and Chester C. Winter, M.D.
11:30-12:00 noon—**Questions.**

Fee: $30.00.

Liver Diseases

Program planned by the University of Southern California School of Medicine

APRIL 27, 28 and 29

Ambassador Hotel and Los Angeles County General Hospital

SUNDAY, APRIL 27

Symposium on Jaundice

(Los Angeles County General Hospital)

2:00-3:00 p.m.—**Panel—Liver Function Tests:**
Telfer B. Reynolds, M.D., Moderator
Charles S. Davidson, M.D.
Bernard J. Haverback, M.D.
Allan G. Redeker, M.D.
3:00-5:00 p.m.—**Clinic on Jaundice.** Small groups will see and discuss interesting patients with the faculty.

MONDAY, APRIL 28

Ambassador Hotel, French Room

9:00-9:40 a.m.—**Viral Hepatitis**—Charles S. Davidson, M.D.
9:40-10:10 a.m.—**Malignant Carcinoid Syndrome**—Bernard J. Haverback, M.D.
10:10-10:40 a.m.—**Wilson's Disease; A Treatable Condition**—Telfer B. Reynolds, M.D.
10:40-10:50 a.m.—**Recess.**
10:50-12:00 noon—**Diagnostic Techniques Applicable to Liver Disease**
Splenoportography—William P. Mikkelsen, M.D.
Hepatic Vein Catheterization—Allan G. Redeker, M.D.
Liver Biopsy—Telfer B. Reynolds, M.D.

TUESDAY, APRIL 29

Ambassador Hotel, French Room

Symposium on Cirrhosis

9:00-9:30 a.m.—**Alcohol vs. Nutrition in the Etiology of Cirrhosis**—Charles S. Davidson, M.D.

Complications of Cirrhosis

9:30-10:00 a.m.—**Ascites**—Telfer B. Reynolds, M.D.
10:00-10:40 a.m.—**Portal Hypertension**—William P. Mikkelsen, M.D., and Telfer B. Reynolds, M.D.
10:40-10:50 a.m.—**Recess.**
10:50-12:00 noon—**Hepatic Coma and Its Relationship to Ammonium Metabolism**—Charles S. Davidson, M.D.

Fee: $30.00. Limited to 100 Registrants.

Postgraduate Courses

WILLIAM A. ALTEMEIER, M.D., Christian R. Holmes Professor of Surgery, University of Cincinnati College of Medicine

CHARLES S. DAVIDSON, M.D., Associate Professor of Medicine, Harvard Medical School, Boston

MARK M. RAVITCH, M.D., Associate Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore

BRONSON S. RAY, M.D., Professor of Clinical Surgery (Chief, Department of Neurosurgery), New York Hospital-Cornell University Medical College, New York City

guest speakers

Management of Trauma

Program planned by College of Medical Evangelists

APRIL 28, 29 and 30

Ambassador Hotel
Embassy Room—Monday and Tuesday
East Venetian Room—Wednesday

MONDAY, APRIL 28

Robert L. Marsh, M.D., Chairman and Moderator

GENERAL TOPIC

Basic Science Applications to Trauma with Particular Attention to Shock and Burns

9:00-9:15 a.m.—**Fluid and Electrolytes**—David B. Hinshaw, M.D.
9:15-9:30 a.m.—**Hemorrhage**—Harry A. Davis, M.D.
9:30-9:45 a.m.—**Vasomotor Responses**—Philip J. Vogel, M.D.
9:45-10:00 a.m.—**Endocrines**—Roy F. Perkins, M.D.
10:00-10:15 a.m.—**Ventilation**—Clarence R. Collier, M.D.
10:15-10:35 a.m.—**Discussion and Summary**—Mark M. Ravitch, M.D.
10:35-10:50 a.m.—**Recess.**
10:50-12:00 noon—**Panel Discussion**
Robert L. Marsh, M.D., Moderator
Mark M. Ravitch, M.D. — Roy F. Perkins, M.D.
Harry A. Davis, M.D. — Philip J. Vogel, M.D.
Clarence R. Collier, M.D. — David B. Hinshaw, M.D.

TUESDAY, APRIL 29

Harry C. Prout, M.D., Chairman and Moderator

GENERAL TOPIC

Injuries to the Thorax and Abdomen

9:00-9:15 a.m.—**Injuries to the Thorax**—Ellsworth E. Wareham, M.D.
9:15-9:30 a.m.—**Injuries to the Abdomen**—Harry C. Prout, M.D.
9:30-9:45 a.m.—**Injuries to Blood Vessels**—Max R. Gaspar, M.D.
9:45-10:00 a.m.—**Injuries to the Genitourinary System**—Roger W. Barnes, M.D.
10:00-10:15 a.m.—**Management of Patients with Burns**—Clifford A. Barber, M.D.
10:15-10:35 a.m.—**Discussion and Summary**—William A. Altemeier, M.D.
10:35-10:50 a.m.—**Recess.**
10:50-12:00 noon—**Panel Discussion:**
Harry C. Prout, M.D., Moderator
William A. Altemeier, M.D.
Roger W. Barnes, M.D. — Clifford A. Barber, M.D.
Max R. Gaspar, M.D. — Ellsworth E. Wareham, M.D.

WEDNESDAY, APRIL 30

Alonzo J. Neufeld, M.D., Chairman and Moderator

GENERAL TOPIC

Injuries to Nervous System, Face, Skeleto-Muscular Structures

9:00-9:20 a.m.—**Injuries to Facial Structures**—Leland R. House, M.D.
9:20-9:40 a.m.—**Mechanism of Cerebral Injuries**—Cyril B. Courville, M.D.
9:40-10:00 a.m.—**Treatment of Injuries to the Nervous System**—Bronson S. Ray, M.D.
10:00-10:20 a.m.—**Injuries to Skeleto-Muscular Structures**—Vernon L. Nickel, M.D.
10:20-10:40 a.m.—**Recess.**
10:40-12:00 noon—**Panel Discussion:**
Alonzo J. Neufeld, M.D., Moderator
Bronson S. Ray, M.D. — Leland R. House, M.D.
Cyril B. Courville, M.D. — Vernon L. Nickel, M.D.

Fee: $30.00.

APPLICATION FORM

POSTGRADUATE ACTIVITIES
CALIFORNIA MEDICAL ASSOCIATION
2975 Wilshire Boulevard
Los Angeles 5, California

Enclosed please find my check for $30.00, made payable to *California Medical Association*. I plan to attend the Postgraduate Course..... in Los Angeles during the California Medical Association annual session.

Name.....

Address.....

☐ I am in General Practice
☐ I limit my practice to.....

Medical school attended.....

Year of graduation.....

Interns and Residents are invited to attend free of charge.

Volunteer Services

The "GEMS" Program

IT IS DIFFICULT to imagine what American life, minus its volunteers, would be like. All of us have been asked to accept responsibility to do volunteer work in some activity important to the health and welfare of our family, of our community, our state or nation. In an address to the Auxiliary, Dr. Edward J. McCormick said, "Every doctor's wife should have a wholesome desire to preserve our democracy and bring continually improved medical care to the American people." The health movement has its finest flowering in this country. Nowhere else in the world has it experienced such an extensive and diversified development. In fact, it has become a permanent and important part of our national health program.

As you know, the Woman's Auxiliary to the California Medical Association does a tremendous volunteer service in nurse recruitment, in the formation of Future Nurse Clubs and in the giving of scholarships; and our public relations projects cover many volunteer services. Recently our Auxiliaries have become interested in the "GEMS" program (good emergency mother substitutes).

Certainly to be a baby sitter is a big challenge. This training is a priceless possession. The National Safety Council is keenly aware of the many parents who hire teenagers for purposes of baby sitting. It felt that for the safety of the baby sitter, the children involved, the parents and the home, there should be a guide for the planning and executing of a community project on safety, with respect to baby sitting. Safety and child care are so interwoven that education on how to care for children must include education in safe practices. Furthermore, so many youngsters in their teens are baby sitting that education must also be directed toward their protection. The formation of a baby sitters council or committee within the local Safety Council organization is basic. Working through existing groups such as the Girl Scouts, Campfire Girls, church youth groups and the P-TA can be of much benefit. All are interested in the welfare and safety of children; all welcome the opportunity to serve the community. Two of our county auxiliaries, Santa Clara and San Bernardino, have this program in operation; both counties feel that they are contributing greatly to the welfare of the community and are serving American medicine as well as providing a much needed service to their friends and neighbors.

The lessons for baby sitter "GEMS" are five in number, at least an hour long—and frequently longer because of group discussion. A packet from the National Safety Council has a suggested outline for a community course, and it covers child care, safety, first aid, what the parents expect, what the baby sitter expects, the prevention and care of accidental poisonings, dealing with aspiration of small articles, burns, and the preparation and serving of meals.

In order to have the course worth while, it must be conducted by persons who are experienced in this subject and who have a respected place in the community. The standards for passing the course are set high so that it is a challenge to the student.

Service to any organization means loyalty to principle and friends. With the "GEMS" program the auxiliary members show that we live what we preach and that we sustain and uphold the high standards of the medical profession.

MRS. LEONARD D. OFFIELD
President, Woman's Auxiliary to the California Medical Association

NEWS & NOTES

NATIONAL • STATE • COUNTY

FRESNO

Dr. Robb Smith, Orange Cove, was elected president-elect of the Fresno County Medical Society at the organization's annual meeting in December, and Dr. John F. Murray, Fresno, was installed as president.

IMPERIAL

Dr. Burke Schoensee of El Centro was elected president of the Imperial County Medical Society at the society's annual meeting in December. He succeeded Dr. A. A. Adames, Holtville. Dr. Robert Chapman of Brawley was elected vice-president and Dr. Ernest Brock, Imperial, secretary-treasurer.

Dr. Milton A. Streuter of Brawley was elected delegate to the California Medical Association.

LOS ANGELES

The International College of Surgeons will hold its eleventh biennial International Congress in Los Angeles March 9 to 14 at the Hotel Ambassador. Surgical sections and general assemblies will present more than 100 national and foreign speakers. A special assembly program on Surgical Emergencies has been arranged for members of the American Academy of General Practice.

A preliminary program may be obtained from Dr. J. M. de los Reyes, general chairman, 1919 Wilshire Boulevard, Los Angeles 57.

* * *

An example of a physician's taking part in nonmedical matters of importance in his local community was given in a page one report by the journal of the Beverly Hills Chamber of Commerce upon the retirement of Dr. James C. Doyle, speaker of the House of Delegates of the California Medical Association, from the chamber's board of directors.

James C. Doyle

That he has followed the recommendation, made by a number of top officials in the American Medical Association, for physicians to take **an active interest in civic and cultural affairs** is shown by the chamber's list of his activities and positions of trust. Dr. Doyle has served as president of the Chamber of Commerce, of the Beverly Hills Men's Club, Beverly Hills Medical Association, Beverly Hills Rotary Club, Beverly Hills Philharmonic Association and Wilshire Boulevard Association; as a director of the Y.M.C.A. and as a director and local chairman of the American Red Cross. Dr. Doyle has given more than 300 lectures, many of them on medical subjects, to nonprofessional audiences.

SACRAMENTO

Dr. Paul Dudley White, professor emeritus of medicine at Harvard University School of Medicine, was the speaker at the first annual medical lecture sponsored by Sacramento County as an expression of the county's appreciation to the 180 physicians who donate their time in care of indigent patients at the Sacramento Hospital.

More than 500 members of the medical and allied professions attended Dr. White's talk on "The Health of the American Family from 1800 to 2000 A.D." The speaker was introduced by Dr. Dwight Murray of Napa, former president of the American Medical Association, who was instrumental in obtaining Dr. White's services.

Sacramento County plans to sponsor similar lectures each year.

SAN FRANCISCO

The April 10 to 12 annual meeting of the **California Tuberculosis and Health Association** has been moved from San Francisco to San Mateo. It will be held at the Villa Hotel, on El Camino Real in San Mateo, instead of at the Sheraton-Palace in San Francisco as originally planned. Meeting jointly with the CTHA will be its medical arm, the California Trudeau Society, the California Conference of Tuberculosis Workers and the California Sanatorium Association.

* * *

The San Francisco Academy of General Practice has announced that the **Fifth Annual Symposia in Medicine** will be given at the Fort Miley Veterans Administration Hospital, 42nd Avenue and Clement Street, on Tuesday evenings for six weeks, starting April 1 and ending May 6, 1958, from 8:00 to 10:00 p.m. The course is given with the cooperation of the medical departments of Stanford University School of Medicine, University of California School of Medicine and the Fort Miley Veterans Administration Hospital, the announcement said.

"The programs are especially formulated to the needs and desires of the general practitioners," the announcement continued, "and every effort is made to stress practical application in the daily practice of medicine."

It is not necessary to be a member of the academy to attend and the resident staffs of any of the hospitals are admitted without fee.

Dr. Forrest M. Willett, associate professor of medicine at Stanford University School of Medicine will act as moderator.

All inquiries and requests for programs should be sent to the chairman, Dr. Alex F. Fraser, 3490 Twentieth Street, San Francisco.

* * *

Establishment of a $210,000 **surgical research fund** at the University of California Medical Center here has been announced by Dr. J. B. deC. M. Saunders, dean of the School of Medicine. The fund will be known as the George K. Rhodes and Wilma Leah Rhodes Research Foundation. It represents the bulk of the estate of the late Dr. Rhodes, who died during World War II service and whose widow died last year.

Income from the fund, Dr. Saunders said, will help support a wide variety of research projects in the department of surgery.

Dr. Rhodes was clinical professor of surgery in the School of Medicine and many years was chief emergency surgeon of the City and County of San Francisco. He had established the bulk of his estate as a trust fund, the principal of which was to be given to the university after he and his wife had died.

At the 1957 annual meeting of the **California Society of Internal Medicine,** held in Palm Springs, the following officers were elected by approximately 225 members who attended: President, Robert L. Smith, Jr., San Francisco; vice-president, Walter P. Martin, Long Beach; secretary-treasurer, Clyde C. Green, Jr., San Francisco; Council members: William L. Cover, San Bernardino; Arthur F. Edwardes, Los Angeles; A. Lee Edgar, San Diego; Edward Shapiro, Beverly Hills; Reginald H. Smart, Los Angeles; James H. Thompson, San Francisco.

The annual meeting will be held October 17 to 19, 1958, in Yosemite Valley.

* * *

Dr. Robert M. Featherstone, who has been professor of pharmacology at the University of Iowa School of Medicine since 1955, has accepted appointment as professor and chairman of the department of pharmacology at the University of California School of Medicine, San Francisco.

Dr. Featherstone succeeds Dr. Hamilton H. Anderson, who had asked to be relieved of his administrative duties.

* * *

A **$2,680,000 research endowment** which will allow Stanford Medical School researchers to pursue unrestricted studies has been established through the estate of Mrs. Sophie K. Hall of Los Angeles.

The bequest, income from which will support the Lendal M. Gray research fund, is the largest single gift from an individual since the founding grant of Senator and Mrs. Leland Stanford in 1885.

Dr. Robert H. Alway, acting dean of the Medical School, said, "The money will be of immense help to us because of its applicability. It will permit us to launch studies in the fields of greatest promise or greatest need."

The grant is named for Mrs. Hall's first husband, a San Francisco shipping executive who was killed in 1911 in an automobile accident. Mr. and Mrs. Gray lived for several years in Atherton, on the northern edge of the Stanford campus.

SANTA CRUZ

Dr. R. Carey Alsberge of Santa Cruz was elected president of the Santa Cruz County Medical Society at the annual meeting in December. He succeeded Dr. James Spencer of Watsonville. Dr. James DePuy of Watsonville was elected vice-president and Dr. William Cress of Santa Cruz, secretary.

Dr. Luther Newhall of Santa Cruz and Dr. Spencer were named delegates to the California Medical Association.

SONOMA

Dr. Lucius L. Button, Santa Rosa, was elected president-elect of the Sonoma County Medical Society at the annual meeting in December, and Dr. Frank E. Lones, Santa Rosa, was installed as president, succeeding Walter E. Webber, also of Santa Rosa. Dr. Clayton Taylor was re-elected secretary-treasurer.

STANISLAUS

The **story of Medicine in Stanislaus County,** as related by many of those who helped write it, featured a recent meeting of the Stanislaus County Medical Society at Del Rio Golf and Country Club.

Discussing facets of county medical history were: Dr. A. M. Roscoe, formerly of Newman and now assistant medical director of Placer County Hospital; Drs. Fred R. DeLappe, E. V. Falk, Earl R. McPheeters, Frank Reardon, Jack Ranson, Ralph Maxwell, Carl Benson, Lloyd D. Mottram, Harold "Brick" Muller, and Herbert McLean Evans, U. C. professor who has achieved international fame. Dr. Evans, a native of Modesto, has instructed many doctors in the county.

Dr. Donald Robertson recounted historical notes and also presented the society with 23 portraits of early day medical figures.

Dr. Evelyn Mayman delivered an eulogy to Dr. C. W. Evans, a prominent figure in medicine in the Stanislaus area.

Dr. Guerne deLappe, outgoing president, was given an inscribed gavel by his successor, Dr. E. K. Prigge. Dr. George Paullus was named president-elect and Dr. John Woolley, secretary-treasurer. Elected to serve on the board of directors were Drs. K. A. Hill and C. Dwight Yates.

Dr. Robert Purvis received a plaque for his three years of service as secretary-treasurer.

Officers and directors presented an engraved trophy to Thomas DeVere, executive secretary of the society.

POSTGRADUATE EDUCATION NOTICES

THIS BULLETIN of the dates of postgraduate education programs and the meetings of various medical organizations in California is supplied by the Committee on Postgraduate Activities of the California Medical Association. In order that they may be listed here, please send communications relating to your future medical or surgical programs to: Mrs. Margaret H. Griffith, Director, Postgraduate Activities, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5.

UNIVERSITY OF CALIFORNIA AT LOS ANGELES

Preventive Psychiatry in Pediatric Practice. Mondays, February 24 to April 28. Ten hours. Fee: $75.00.

Recent Advances in General Surgery. Wednesday, February 26 to June 16. Twenty-six hours. Fee $75.00.

Postgraduate Medical Lecture Series (Saint Mary's Hospital, Long Beach). Tuesdays, March 4 to May 20. Twenty-four hours. Fee: $50.00.

Arthritis—Practical Considerations in Diagnosis and Management. Wednesday, March 5. Six hours. Fee: $5.00.

Diagnostic Parasitology for Laboratory Technologists. Wednesdays, March 12 to May 28. Thirty-six hours. Fee: $40.00.

The Clinical Approach to Diseases of the Blood. Wednesday, March 19. Six hours. Fee: $25.00.

Proctology. Wednesday, April 16. Six hours.†

Symposium—Occupational Therapists and Related Groups. Saturday, April 26. Six hours.†

†Fee to be announced.

Emotional Problems in Office Practice (University of California Residential Conference Center, Lake Arrowhead). Thursday through Saturday, June 5 to 7. Twelve hours.†

Collagen Diseases. Friday and Saturday, June 6 and 7. Nine hours. Fee: $35.00.

Seventh Annual Symposium on Medical Technology. Saturday and Sunday, June 14 and 15. Twelve hours. Fee: $20.00.

Techniques of Hypnosis (Limited to 40). Monday through Wednesday, June 16 to 18. Fifteen hours.†

Advanced Techniques and Application of Hypnosis (Limited to 25). Wednesday through Friday, June 18 to 20. Fifteen hours.†

Techniques of Surgery. Monday through Friday (two weeks), July 21 to August 1. Seventy hours. Fee: $500.00.

Internal Medicine (at University of California Residential Conference Center, Lake Arrowhead), Wednesday through Saturday, August 20 to 23. Twelve hours.†

Surgical Anatomy (Dissection). Friday and Saturday. Date to be announced. Six hours.†

Obstetrics and Gynecology. Date, hours and fee to be announced.

Surgery of Trauma. Date, hours and fee to be announced.

Casts, Splints and Bandages. Date to be announced. Wednesday. Six hours.†

Contact: Thomas H. Sternberg, M.D., Assistant Dean for Postgraduate Medical Education, U.C.L.A., Los Angeles 24. BRadshaw 2-8911, Ext. 202.

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

Postgraduate Seminars in Pharmacy. Thursday to Saturday, February 20 to 22. Fifteen hours. Fee: $25.00.

Medical Emergencies. (Herrick Hospital, Berkeley). Thursday through Saturday, February 20, 21 and 22. Twenty hours. Fee: $50.00.

Course for Physicians in General Practice. Monday through Friday, February 24 to 28, 1958. Thirty-two hours.†

Bedside Cardiology. Monday through Friday, March 10 through 14. Thirty-five hours.†

Children's Hospital Medical Seminar (Allergy). Saturday, March 29. Seven hours.†

Ear-Nose-Throat. Friday and Saturday, June 6 and 7. Fourteen hours. Fee: $40.00.

Diagnostic Radiology. Date, hours and fee to be announced.

Interpretation of Laboratory Tests. Date, hours and fee to be announced.

Fundamental Principles of Radioactivity and the Diagnostic and Therapeutic Uses of Radioisotopes. Two or three month course limited to one enrollee per month. Per month, $250.00.

Contact: Seymour M. Farber, M.D., Head, Postgraduate Instruction, Office of Medical Extension, University of California Medical Center, San Francisco 22. MOntrose 4-3600, Ext. 665.

STANFORD UNIVERSITY SCHOOL OF MEDICINE

Morning Clinical Conferences, each Monday, Room 515. *Contact:* D. H. Pischel, M.D., Professor, Division of Ophthalmology, Stanford University School of Medicine, 2398 Sacramento St., San Francisco 15.

Postgraduate Conference in the Surgery of Trauma. March 24 to 28.

Postgraduate Conference in Ophthalmology. March 31 to April 4.

UNIVERSITY OF SOUTHERN CALIFORNIA, LOS ANGELES

Cardiac Resuscitation. Sponsored by the Los Angeles County Heart Association each Wednesday throughout the year, 4 to 6 p.m. Residents admitted without fee. Tuition for all other physicians: $30.00. (Each session all-inclusive.)

Basic Home Course in Electrocardiography. One year Postgraduate Series, electrocardiogram interpretation by mail. Physicians may register at any time and receive all 52 issues. Fifty-two weeks. Fee: $100.00.

Advance Home Course in Electrocardiography. One year postgraduate series, electrocardiogram interpretation by mail. Fifty-two issues: $85.00. Physicians may register at any time.

Office Orthopedics. Designed to be of value to the physician in general practice. March 14 to 16, Hotel Statler, Los Angeles, 9 to 5 each day.

The Edematous State. March 28 and 29, Hotel Statler, Los Angeles, 9 to 5.

Special Announcement: From August 5 to August 21, 1958, the University of Southern California School of Medicine will hold a postgraduate course in Honolulu and on board the *S.S. Matsonia.* The course will center around actual case histories, which will be used to emphasize diagnostic and therapeutic features.

Contact: Phil R. Manning, M.D., Director, Postgraduate Division, University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 33. CApital 5-1511.

COLLEGE OF MEDICAL EVANGELISTS

Vue-Vox Postgraduate Refresher Courses. Courses are made up of four or more half-hour lectures each, recorded on hi-fi magnetic tape and illustrated by 35-mm. filmstrips or slides in full color, and adapted for use on any standard tape recorder and filmstrip or slide projector, automatic or manual.

Contact: Paul D. Foster, M.D., chairman, Committee on Audio-Visual Courses, College of Medical Evangelists School of Medicine, 316 North Bailey St., Los Angeles 33.

CALIFORNIA MEDICAL ASSOCIATION POSTGRADUATE COURSES

POSTGRADUATE INSTITUTES—1958

SAN BERNARDINO, RIVERSIDE AND ORANGE COUNTIES, in cooperation with College of Medical Evangelists, Arrowhead Springs Hotel, San Bernardino County, February 13 and 14, 1958. Chairman: Elmer O. Carlson, M.D., 756 N. Euclid Ave., Ontario.

WEST COAST COUNTIES, in cooperation with UCLA School of Medicine, Golden Bough Theater and La Playa Hotel, Carmel, March 6 and 7, 1958. Chairman: Howard C. Miles, M.D., 535 E. Romie Lane, Salinas.

SACRAMENTO VALLEY COUNTIES, in cooperation with University of California School of Medicine. El Dorado Hotel. March 14 and 15. Chairman: Fred Schroeder, M.D., 2909 J Street, Sacramento.

SAN JOAQUIN VALLEY COUNTIES, in cooperation with Stanford University School of Medicine, Hotel Californian, Fresno, March 20 and 21, 1958. Chairman: Henry L. Tiesche, M.D., 1759 Fulton St., Fresno.

NORTH COAST COUNTIES, in cooperation with USC School of Medicine, Hoberg's Resort, Lake County, April 10, 11 and 12, 1958. Chairman: Alfred A. Thurlow, Jr., M.D., 185 Sotoyome Ave., Santa Rosa.

Contact: One of the chairmen listed above, or Mrs. Margaret H. Griffith, Director, Postgraduate Activities, California Medical Association, 2975 Wilshire Blvd., Los Angeles 5. DUnkirk 5-2341.

* * *

AUDIO DIGEST FOUNDATION, a nonprofit subsidiary of the C.M.A., now offers (on a subscription basis) a series of hour-long tape recordings designed to keep the physician abreast of current happenings in his particular field. Composed of practice-useful abstracts from 600 leading journals, with short lectures and editorial comments from prominent physicians, Audio Digest offers programs covering general practice, surgery, internal medicine, obstetrics and gynecology, and pediatrics.

Contact: Claron L. Oakley, editor, 1919 Wilshire Blvd., Los Angeles 57.

Medical Dates Bulletin

FEBRUARY MEETINGS

COLORADO STATE MEDICAL SOCIETY Midwinter Clinical Session, February 18 to 21, Shirley-Savoy Hotel, Denver. *Contact:* Harvey T. Sethman, executive secretary, 835 Republic Bldg., Denver 2.

CITY OF HOPE MEDICAL CENTER Symposium on "Effects of Drugs on the Central Nervous System," City of Hope Medical Center, Duarte, February 19 to 21. Fee is $10 for one day and $25 for full symposium. *Contact:* Julian Love, M.D., director, School of Postgraduate Medicine, City of Hope Medical Center, Duarte.

CALIFORNIA BLOOD BANK Annual Meeting, Miramar Hotel, Santa Barbara, February 20 to 22. *Contact:* Helen E. Drew, secretary, P. O. Box 1148, Santa Barbara.

LOS ANGELES RADIOLOGICAL SOCIETY Tenth Annual Midwinter Conference, Biltmore Hotel, Los Angeles, February 22 and 23, 9:30 a.m. *Contact:* John H. Eaton, M.D., secretary-treasurer, 65 N. Madison Ave., Pasadena 1.

HIGHLAND-ALAMEDA COUNTY HOSPITAL Symposium: Chemistry, Biochemistry and Metabolism of Lipids, at the hospital. 9:00 a.m. to 5:00 p.m., February 24 through 28; 9:00 a.m. to 12:00 noon on March 1. Auspices will be "Center for Post-Doctoral Study" and "The Institute for Metabolic Research." *Contact:* L. W. Kinsell, M.D., director of the Institute for Metabolic Research, 2701 14th Avenue, Oakland 6.

MARCH MEETINGS

INTERNATIONAL COLLEGE OF SURGEONS 23rd Annual Congress of U. S. and Canadian Sections 11th Biennial International Congress, March 9 to 13, Hotel Ambassador, Los Angeles. *Contact:* J. M. de los Reyes, M.D., general chairman, 2010 Wilshire Blvd., Los Angeles 57.

SOUTHWESTERN PEDIATRIC SOCIETY Annual Lecture Series, March 11 and 12, Statler Hotel, Los Angeles. *Contact:* D. Hovsepian, M.D., program chairman, 960 E. Green St., Pasadena.

CALIFORNIA SOCIETY OF PLASTIC SURGEONS Annual Meeting, March 20 to 22, Del Monte Lodge, Del Monte, California. *Contact:* Benjamin F. Edwards, M.D., 2200 Santa Monica Blvd., Santa Monica.

AMERICAN COLLEGE HEALTH ASSOCIATION Annual Meeting, March 25 to 29, Ambassador Hotel, Los Angeles. *Contact:* Donald McKinnon, M.D., Student Health Service, University of California Medical Center, Los Angeles 24.

APRIL MEETINGS

SAN FRANCISCO HEART ASSOCIATION Nurses Institute on the Care of the Cardiac Patient, April 2 and 3. Marina Junior High School, San Francisco, 8:30 a.m. to 4:00 p.m. *Contact:* Miss Jean Sullivan, program director, 259 Geary St., San Francisco.

CALIFORNIA TUBERCULOSIS AND HEALTH ASSOCIATION, CALIFORNIA TRUDEAU SOCIETY and the CALIFORNIA CONFERENCE OF TUBERCULOSIS WORKERS Annual Meeting, Villa Hotel, San Mateo, April 9 through 12, 1958. *Contact:* E. L. Daggett, director public information, 130 Hayes Street, San Francisco 2, HEmlock 1-8771.

VALLEY CHILDREN'S HOSPITAL Spring Clinics, April 11 and 12. Roosevelt High School Auditorium, Fresno. *Contact:* Valley Children's Hospital, Fresno.

AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS, April 21 to 23, Los Angeles. *Contact:* John C. Ullery, M.D., secretary, 15 South Clark St., Chicago 3.

CALIFORNIA MEDICAL ASSOCIATION Annual Meeting, Ambassador Hotel, Los Angeles, April 27 to 30. *Contact:* John Hunton, executive secretary, 450 Sutter St., San Francisco 8, or Ed Clancy, director, Public Relations, 2975 Wilshire Blvd., Los Angeles 5.

MAY MEETINGS

LOS ANGELES COUNTY HEART ASSOCIATION Annual Membership Meeting, May 1, 7:00 p.m., Los Angeles. *Contact:* Chauncey A. Alexander, executive director, 660 S. Western Avenue, Los Angeles 5.

WESTERN GERONTOLOGICAL SOCIETY Third Annual Convention, May 3 and 4, Hotel Statler, Los Angeles. Begins 1:00 p.m. May 3. *Contact:* Chas. A. Preuss, M.D., president, 1317 Santa Barbara Street, Santa Barbara.

AMERICAN PSYCHIATRIC ASSOCIATION Annual Meeting, May 12 to 16, Civic Auditorium, San Francisco. *Contact:* Alfred Auerback, M.D., 450 Sutter St., San Francisco.

HAWAIIAN DIVISIONAL MEETING, AMERICAN PSYCHIATRIC ASSOCIATION, May 17 to 22, Hawaiian Village, Honolulu. *Contact:* Alfred Auerback, M.D., 450 Sutter St., San Francisco.

WESTERN BRANCH, AMERICAN PUBLIC HEALTH ASSOCIATION Annual Meeting with Canadian Public Health Association, May 18 to 23, Vancouver, B. C. *Contact:* Mrs. L. Amy Darter, secretary-treasurer, 2151 Berkeley Way, Berkeley 4.

AMERICAN LARYNGOLOGICAL ASSOCIATION, May 19 to 20, San Francisco. *Contact:* James H. Maxwell, M.D., secretary, University Hospital, Ann Arbor, Michigan.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION, May 21 to 23, San Francisco. *Contact:* F. Johnson Putney, M.D., secretary, 1719 Rittenhouse Square, Philadelphia, Pa.

CALIFORNIA HEART ASSOCIATION Annual Meeting, Scientific Session and Directors Meeting, Hacienda Motel, Fresno, May 23 to 25. *Contact:* J. Keith Thwaites, executive director, 1428 Bush St., San Francisco.

SUMMER AND FALL MEETINGS

AMERICAN COLLEGE OF CHEST PHYSICIANS 24th Annual Meeting, June 18 to 22, San Francisco. *Contact:* Mr. Murray Kornfeld, executive director, 112 East Chestnut St., Chicago 11, Ill.

AMERICAN MEDICAL ASSOCIATION Annual Meeting, June 23 to 27, San Francisco. *Contact:* American Medical Association, 535 North Dearborn St., Chicago 10.

AMERICAN PROCTOLOGIC SOCIETY, June 29 to July 3, Los Angeles. *Contact:* Norman D. Nigro, M.D., secretary, 10 Peterboro St., Detroit 1, Michigan.

IDAHO STATE MEDICAL ASSOCIATION Annual Convention, July 6 to 9, Sun Valley, Idaho. *Contact:* Armand L. Bird, executive secretary, 364 Sonna Building, Boise.

WASHINGTON STATE MEDICAL ASSOCIATION Annual Convention, September 14 to 17, 1958, Spokane, Washington. *Contact:* Ralph W. Neill, executive secretary, 1309 Seventh Ave., Seattle, Wash.

CALIFORNIA ACADEMY OF GENERAL PRACTICE Tenth Annual Scientific Assembly, October 5 to 8, San Francisco. *Contact:* William W. Rogers, executive secretary, 461 Market Street, San Francisco 5.

CALIFORNIA SOCIETY OF INTERNAL MEDICINE Annual Meeting, October 17 to 19, Ahwahnee Hotel, Yosemite. *Contact:* Mrs. Mildred B. Coleman, executive secretary, or Dr. Clyde C. Greene, secretary-treasurer, 350 Post St., San Francisco 8.

AMERICAN HEART ASSOCIATION Scientific Sessions and Meetings, October 24 to 27, Fairmont Hotel and Civic Auditorium, San Francisco. *Contact:* J. Keith Thwaites, executive director, California Heart Association, 1428 Bush Street, San Francisco 9.

For Your Patients—

A Personal Message to YOU:

As your personal physician I consider it both a privilege and a matter of duty to be available in case of an emergency. But, being only human you can understand that there are times when I may not be on call. I might be at a medical meeting outside the city, on a bit of a vacation—or even ill.

Consequently, I thought it would be a good precaution if—on this gummed paper which you can paste in your telephone book or in your medicine cabinet—I listed numbers where I can be reached at all times. Also, the number of a capable associate as an added service. Here they are:

_____	_____	_____
OFFICE	HOME	MY DOCTOR
_____	_____	_____
OFFICE	HOME	ASSOCIATE

Sincerely,

_____, M.D.

MESSAGE NO. 1. Attractive, postcard-size leaflets printed on gummed paper, you to fill in telephone numbers and your signature. Available in any quantity, at no charge, as another service to CMA members. Please order by Message Number from CMA, PR Department, 450 Sutter, San Francisco.

EIGHTH ANNUAL REGIONAL

1 WEST COAST COUNTIES

Presented by Postgraduate Activities Committee of the California Medical Association, in cooperation with Monterey, Santa Cruz and San Benito County Medical Societies, and the Postgraduate Division of University of California, Los Angeles, Thomas H. Sternberg, M.D., Assistant Dean for Postgraduate Medical Education.

Golden Bough Theater and

La Playa Hotel, Carmel... March 6 and 7

PROGRAM

THURSDAY, MARCH 6, 1958

9:00-9:35 a.m.—**Recent Advances in Urology**—Willard E. Goodwin, M.D.

9:35-10:10 a.m.—**New Adenovirus Infections in Children**—John M. Adams, M.D.

10:25-11:00 a.m.—**Diagnosis and Management of Anemia**—William N. Valentine, M.D.

11:00-12:00 a.m.—Three Panel Discussions (you may go to one of your choice):

Enuresis
Bleeding Disorders
Psychology of Malpractice

12:00-2:00 p.m.—Luncheon Meeting, La Playa Hotel

New Trends in Postgraduate Education—Edward C. Rosenow, Jr., M.D., Chairman, California Medical Association, Postgraduate Activities Committee

Open question and answer session on morning lectures

2:00-2:35 p.m.—**Abdominal Pain**—Willard E. Goodwin, M.D.

2:35-3:10 p.m.—**Hepatitis**—John C. Sharpe, M.D.

3:25-4:00 p.m.—**Emotional Problems**—Frank F. Tallman, M.D.

4:00-5:00 p.m.—Two Panel Discussions (you may go to one of your choice):

Emotional Problems
Management of the Obstructing Prostate in the Poor Risk Patient

6:30 p.m.—No Host Social Hour, La Playa Hotel

8:00 p.m.—Dinner. Monterey Peninsula Country Club in Del Monte Forest

The Use of Hypnosis in Medicine—Roy M. Dorcus, Ph.D.

FRIDAY, MARCH 7, 1958

9:00-9:35 a.m.—**Diagnostic Application of Radioisotopes**—Leslie R. Bennett, M.D.

9:35-10:10 a.m.—**Diagnosis and Management of Skin Tumors**—Thomas H. Sternberg, M.D.

10:25-11:00 a.m.—**Surgical Treatment of Superficial Lesions**—Franklin L. Ashley, M.D.

11:00-12:00 a.m.—Three Panel Discussions (you may go to one of your choice):

Skin Tumor Clinic
Diagnosis and Treatment of Malignant Blood Disorders
Psychology of Malpractice

12:00-2:00 p.m.—Luncheon Meeting, La Playa Hotel

Open question and answer session on morning lectures

2:00-2:35 p.m.—**Examination and Primary Treatment of Hand Injuries**—James N. Wilson, M.D.

2:35-3:10 p.m.—**Emotional Problems**—Floyd M. Estess, M.D.

3:25-4:00 p.m.—**Newer Therapy in Diabetes**—Josiah Brown, M.D.

4:00-5:00 p.m.—Three Panel Discussions (you may go to one of your choice):

Emotional Problems
Treatment of Thyroid Disease
Hand Injuries

HOST: Monterey County Medical Society . . . **REGIONAL CHAIRMAN:** Howard C. Miles, M.D., 535 East Romie Lane, Salinas, California . . . **Institute Fee:** $20.00* For additional information, contact Postgraduate Activities office, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5. **All California Medical Association members and their families are cordially invited to attend.**

*$10.00 for physicians who have registered at a previous Institute in Spring, 1958.

POSTGRADUATE INSTITUTES

2 SACRAMENTO VALLEY COUNTIES

Presented by Postgraduate Activities Committee of the California Medical Association, in cooperation with Amador, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas, Sacramento, San Joaquin-Calaveras, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo, Yuba, Del Norte, Humboldt, Napa, Mendocino-Lake, Solano, Sonoma, Marin and Contra Costa County Medical Societies, and the University of California, San Francisco, Seymour M. Farber, M.D., Head of Medical Extension.

El Dorado Hotel, Sacramento
March 14 and 15

PROGRAM

FRIDAY, MARCH 14, 1958

MORNING SESSION

The Desire to Live—The Younger Citizen

9:00-9:45 a.m.—**Genetics**—To be announced.

9:45-10:30 a.m.—**Differential Diagnosis and Management of Childhood Fevers**—Edward B. Shaw, M.D.

10:45-11:30 a.m.—**Differential Diagnosis of Surgical Problems**—Leon Goldman, M.D.

11:30-12:15 p.m.—**Behavior Problems in Children**—George H. Schade, M.D.

12:15-1:50 p.m.—Luncheon
Panel: **Behavior Problems and their Management**

AFTERNOON SESSION

Learning to Live—The Citizen in the Middle Years

2:00-2:45 p.m.—**Fads and Fancies in Arteriosclerosis**—Richard J. Havel, M.D.

2:45-3:30 p.m.—**Nutritional Aspects of Liver Disease**—John V. Carbone, M.D.

3:45-4:30 p.m.—**Endocrine Changes Influencing Gynecological Problems**—Alan Margolis, M.D.

4:30-5:15 p.m.—**Conflicts and Concerns of the Middle Years**—August R. Prestwood, M.D.

No Host Social Hour—El Dorado Lounge

SATURDAY, MARCH 15, 1958

MORNING SESSION

Appreciative Living—The Citizen in the Aging Years

9:00-9:45 a.m.—**Endocrine Changes with Aging**—Gilbert S. Gordan, Jr., M.D., Ph.D.

9:45-10:30 a.m.—**Surgical Procedures: Indications and Contraindications**—Robert C. Combs, M.D.

10:45-11:30 a.m.—**Aids from Physical Medicine**—Gregory Bard, M.D.

11:30-12:15 p.m.—**Psychiatric Aspects of the Aging Mind**—Joseph B. Wheelwright, M.D.

12:15-1:50 p.m.—Luncheon
Panel: **Management of Traumatic Situations in the Elder Citizen**

AFTERNOON SESSION

The Neglected Business World of the Physician

2:00-2:30 p.m.—**Clinical Pathological Conference on Tax Problems of the Medical Profession**—Mr. Clarence E. Musto

2:30-3:00 p.m.—**Financial Planning for Physicians**—Mr. David A. Bardes

3:00-3:30 p.m.—**A Protective Investment Program—Tax Free Bonds, Common Stocks, Mutual Funds**—Mr. Albert E. Schwabacher, Jr.

3:45-5:00 p.m.—Panel Discussion and Questions from the Audience:
Dynamic Business Concepts in Planning for the Future

HOST: Sacramento Society for Medical Improvement . . . **REGIONAL CHAIRMAN:** Fred Schroeder, M.D., 2909 J Street, Sacramento, California . . . **Institute Fee:** $20.00* For additional information, contact Postgraduate Activities office, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5. **All California Medical Association members and their families are cordially invited to attend.**

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3 SAN JOAQUIN VALLEY COUNTIES

Presented by Postgraduate Activities Committee of the California Medical Association, in cooperation with Fresno, Inyo-Mono, Kern, Kings, Madera, Merced-Mariposa, San Joaquin, Stanislaus, Tulare and Tuolumne County Medical Societies, and the Stanford University School of Medicine, Lowell A. Rantz, M.D., Director of Postgraduate Programs.

Hotel Californian, Fresno... March 20 and 21

PROGRAM

THURSDAY, MARCH 20, 1958

9:00-9:40 a.m.—**Hospital Acquired Infection**—Lowell A. Rantz, M.D.

9:40-10:20 a.m.—**Treatment of Bleeding Esophageal Varices**—Roy B. Cohn, M.D.

10:30-11:15 a.m.—**The Role of the Artificial Kidney in Treatment**—John Young, M.D.

11:15-12:00 noon—**The Economic Outlook and the Physician's Investment Program**—Mr. Hood M. Harris

12:00-2:00 p.m.—Luncheon. Question and Answer Session on **Physician's Investment Program**

2:00-3:00 p.m.—**Treatment of Common Fractures**—Donald E. King, M.D.

3:00-5:00 p.m.—Three Panel Discussions (you may go to one of your choice):

Management of Highway Injuries

Treatment of Hypertension

Hematological Problems in the First Year of Life

FRIDAY, MARCH 21, 1958

9:00-10:00 a.m.—**Screening Methods in Diagnosis of Cancer**—Laurens P. White, M.D.

10:00-12:00 noon—Three Panel Discussions (you may go to one of your choice):

Obstetrical Emergencies

Diagnosis and Treatment of Jaundice

Medical Diseases of the Kidney

12:00-2:00 p.m.—Luncheon

2:00-3:00 p.m.—**Diagnostic and Therapeutic Use of Isotopes**—Joseph P. Kriss, M.D.

3:00-5:00 p.m.—Three Panel Discussions (you may go to one of your choice):

Hematology

Problems of Surgery and Pregnancy in Patients with Heart Disease

Hemoptysis, Hematemesis and Melena

HOST: Fresno County Medical Society . . . **REGIONAL CHAIRMAN:** Henry L. Tieche, M.D., 1759 Fulton Street, Fresno, California . . . **Institute Fee: $20.00*.** For additional information, contact Postgraduate Activities office, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5. **All California Medical Association members and their families are cordially invited to attend.**

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THE PHYSICIAN'S *Bookshelf*

MEDICAL PHYSIOLOGY—Tenth Edition—Edited by Philip Bard, Professor of Physiology, School of Medicine, The Johns Hopkins University. The C. V. Mosby Company, St. Louis, 1956. 1421 pages, $14.00.

It is almost customary for textbooks in physiology to have a heavy emphasis in one field with rather cursory treatments of other fields. In the case of Bard's *Medical Physiology* the reverse seems to be true. In general the treatment of the subject matter of physiology shows rare balance. Circulation justly receives the most extensive treatment, but not to the detriment of respiration, alimentation, metabolism and nutrition, endocrinology and neurophysiology. Each of the above are dealt with in a thorough, business-like manner. Perhaps special mention should be made of the chapters on the circulation because of their easy readability and, indeed, beauty of prose styling. The principal defect in the balance of the book is its inadequate treatment of the kidney. Less space is devoted to the entire subject of kidney function than to the reflex activity of the spinal cord. However, although this is a serious defect in any book intended for use with medical students, it is compensated for to some extent by the treatment of subjects of clinical importance that are usually neglected. For example, in the section entitled "Respiration" alone the chapters on "Abnormal types of breathing," "Anoxia," and the "Poisonous effects of nitrogen, oxygen and carbon dioxide" contain much physiological pathology frequently not introduced in medical education until later in the clinical years and hence often not considered "physiological" by the student.

With the exception noted above this text is well-rounded, easy to read and quite complete. It should receive wide acceptance as a basic reference as well as teaching text in medical physiology.

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EXPERIMENTAL PSYCHOPATHOLOGY — Edited by Paul H. Hock, M.D., New York State Psychiatric Institute; College of Physicians and Surgeons, Columbia University, New York City; and Joseph Zubin, Ph.D., New York State Psychiatric Institute; Department of Psychology, Columbia University. The Proceedings of the 45th Annual Meeting of the American Psychopathological Association, held in New York City, June 1955. Grune & Stratton, New York, 1957. 275 pages, $6.50.

This collection of papers from the 1955 meeting of the American Psychopathological Association is designed to present the main experimental trends in the field of psychopathology. In the words of the editors: "It covers the entire gamut from animal to man, from perception of social attitudes to experimental induction of depersonalization." The papers are evidence of the increasing attempts to apply the scientific method to the study of emotional disorders—and to test psychogenic hypotheses.

Some of the work on experimental animals, while interesting, will be difficult for the physician, except those familiar with the language of experimental psychology, to understand. However approach-avoidance experiments performed with cats by Norma A. Scheflen, Ph.D., demonstrated how fear produced in one situation was carried over to a similar situation and how extinction of fear in the similar situation reduced the fear of the original situation. A possible relationship between such experimental findings and the development and treatment of psychiatric disorders was suggested.

Drs. Gantt and Dykman, in conditioning experiments with dogs, demonstrated an increase in heart rate upon application of the stimulus that appeared before somatic motor conditioned responses and that persisted for long periods of time and sometimes accompanied by the development of additional "neurotic" symptoms after the experiments were discontinued. Some inferences regarding cardiac neuroses in humans were suggested.

Other papers dealt with behavior and metabolic cycles found in man and produced experimentally in animals by brain or endocrine gland lesions or by severe stress, with the effects of hallucinogenic drugs, with psychotherapy, studies in human ecology and the problem of "consciousness" and awareness.

The volume will appeal mostly to the physician who is concerned with the investigation or treatment of emotional disorders but will be of interest to others who are following the changing trends in psychiatry.

NORMAN Q. BRILL, M.D.

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STEPS FOR TODAY TOWARD BETTER MENTAL HEALTH—Pointers to Action for All Who Give Health Services—Report of the 1957 National Health Forum. Edited by Josephine Nelson and Elizabeth M. Dach. National Health Council, 1790 Broadway, New York 19, N. Y., 1957. 118 pages, $1.50.

The National Health Forum annually considers a health problem or development of nationwide concern needing planning and perhaps action. In 1957 the Forum focused on "Steps for Today Toward Better Mental Health." This summary provides an excellent manual for individuals or groups interested in the Community Mental Health services program.

One interesting section "*When to Call the Psychiatrist*" lists the following as bases for seeking psychiatric help: (1) When a person is not himself for a period of time. (2) When a person expresses ideas which you know don't make sense. (3) When a person makes silly mistakes over a period of time and you know that he knows better. (4) When a person acts or talks like he doesn't know where he is or what day it is, for a period of time. (5) When one consistently makes those around him unhappy, upset, worried, over a period of time, say a few months or so, and there is no reasonable, acceptable explanation that is not rationalization. (6) When a person drinks enough to interfere with work, school, or decent family or social relationships. (7) When a person loses appetite or weight over a month or so, and there is no physical illness or need to lose weight. (8) When one has trouble sleeping over a period of time, losing sleep to the point that he shows irritability.

PRACTICAL USE OF THE OFFICE LABORATORY AND X-RAY — Including the Electrocardiograph — Paul Williamson, M.D. The C. V. Mosby Company, St. Louis, 1957. 323 pages, $10.75.

This manual attempts to encompass laboratory, ECG and radiologic procedures for the office. As a laboratory guide to the clinician, the work may have merit, but for the technician, a constant compromise between limitations of the small laboratory and the need for up-to-date methods is evident.

After rather extensive discussions of technique in hematology, bacteriology, chemistry, some parasitology, mycology, and immunology, the author states, "I would recommend that the physician train his own technician, since school-trained technicians are experts in advanced techniques." With the decreasing exposure of physicians to the laboratory in present graduate training, one might well question the ability of most physicians, young or old, to train a "technician," even for the "office laboratory." The techniques a student can pick up from a well-trained technologist make it preferable to send the former to a good medical laboratory for a more satisfactory introduction.

Although a discussion of errors in technique is given for the procedures presented, some precautions should be added, such as use of disposable lancets for obtaining blood (to reduce serum hepatitis), and care in injecting BSP dye, since leakage around the vein can be very distressing. No mention is made of test tapes or sticks although these have largely replaced Benedict's solution as a qualitative test, and are time-saving for the small laboratory. Other tablet or stick tests for urinalysis are also omitted. The entire technical discussion of chemical tests is omitted with the explanation that instructions are given in the manual accompanying a photocolorimeter.

A section on Electrocardiography discusses some of the indications for use of the ECG, points of interpretation, and possible errors. In the diagnosis of "The Coronary Attack" the use of the transaminase determination is omitted. Regarding the difficult cardiac case the author properly concludes ". . . it is probably best to refer the ECG *and the patient* to a cardiologist for diagnosis and recommendations."

The radiology section outlines the diagnosis and to a certain extent the differential diagnosis of certain normal and abnormal findings, in many instances with line drawings. The author displays great faith in the x-ray diagnosis of certain conditions, such as emphysema. Concerning this he states, "Errors in (x-ray) diagnosis are rare."

Similarly, concerning cardiac radiology he writes, "It is of course easy to tell both clinically and by x-ray what chambers of the heart are enlarged." The correct diagnosis of enlargements of the ventricle, especially the right ventricle, are often quite difficult to determine. In connection with the diagnosis of intra-atrial septal defects (the author doubtless means inter-atrial) he writes, "Errors are practically unheard of in diagnosing this condition. Diagnosis is simple and apparent in most cases, both clinically and roentgenologically. This is not true when very young babies are studied by x-ray, but it holds for adults." This is indeed the height of optimism.

The instructions for barium enema preparations are as follows: "The patient should eat lightly the day before examination and not take a laxative. In the evening before retiring he should take a plain water enema and upon rising the next morning take at least four plain water enemas. If the return from the fourth enema is not clear, he should take still another." This will tax the elderly and yield poorer results than oleum ricini. For the diagnosis of simple colitis, the author apparently relies on finding a portion of the colon that shows spasm and loss of normal markings. The author then saves the day by noting that an irritable colon must be diagnosed clinically, not roentgenologically.

The technique and interpretation of hysterosalpingography and other contrast media procedures are outlined, but nowhere is there mention of the hazards to the physician or his patient from excessive amounts of diagnostic radiation.

For the office laboratory "technician," introduction to technique by a well-trained technologist would seem more satisfactory than the use of a text which, by the author's admission, is "a beginning—and nothing more." And for the busy clinician, the help and advice of a laboratory consultant, electrocardiologist or radiologist in setting up whatever procedures are considered desirable, will in most cases be a saving of time and money.

C. M. McCandless, M.D.
L. H. Garland, M.B.

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ROENTGEN DIAGNOSIS OF ABDOMINAL TUMORS IN CHILDHOOD—Charles M. Nice, Jr., M.D., Ph.D., Alexander R. Margulis, M.D., and Leo G. Rigler, M.D., Department of Radiology, University of Minnesota Medical School. Charles C. Thomas, Publisher, Springfield, Illinois, 1957. 75 pages, $4.00.

This monograph is divided into eight sections, the major ones dealing with anatomic and embryologic factors, and a series of roentgen considerations. Masses in the region of the liver and other intraperitoneal sites are first considered. Then renal and adrenal masses. Then various other extraperitoneal masses are described in a series of four chapters.

The roentgen diagnosis of certain of these tumors is well outlined. However, the method of presentation might be a little more systematic, and in the next edition it would be worthwhile adding material on differential diagnosis. The importance of gonadal protection in the examination of infants and children does not receive the emphasis which it should. The work will be of value to medical students, especially those in pediatrics. The authors are on the staff of the University of Minnesota Department of Radiology, and all very competent in their field.

L. H. Garland, M.B.

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DYNAMIC PSYCHIATRY IN SIMPLE TERMS—Robert R. Mexer, M.D., Senior Staff Psychiatrist, Community Clinic, Massachusetts Mental Health Center and Harvard Medical School. Springer Publishing Company, Inc., 44 East 23rd St., New York 10, N. Y., 1956. 174 pages, $2.50.

The growing interest in the application of psychologic knowledge in general medical practice suggests a very real need for a book such as this one.

In general, it is felt that this book very well fulfills this need. Here is presented briefly but clearly a concept of personality development which is followed by descriptions of the various psychiatric disorders in the light of this theory. The valuable material on meeting the psychiatric patient, as well as a concluding chapter on "The Normal Life," add to the value of the book.

Unquestionably, there are psychiatrists who would differ with individual parts of the book. In the main, however, the outline of development, the classification of illnesses and recommendations for care of the patient are those that are in common use today. Perhaps most importantly, the suggestions for dealing with various symptom combinations and illnesses are made most clear. It is in this area that the individual practitioner will find the book of value. It is important, however, to remember that treatment in psychiatric conditions depends as much on good knowledge of personality structure and psychopathology, as general medicine does on physiology and general pathology. This book does a real service by bringing all of these factors together in an organized and clear fashion.

Henry H. Work, M.D.